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FOURTEEN CASES OF CARCINOMA OF THE MIDDLE LOBE BRONCHUS

by

J. Schvarcz

The right middle lobe of the lung has generally been regarded as a relatively rare situation for carcinoma. On the other hand, attention has been focused during recent years on an increasing number of cases of middle lobe syndrome. In the course of the last 5 years we have had the opportunity of investigating several cases of carcinoma of the middle lobe roentgenologically, a number of these having been primarily interpreted as being cases of middle lobe syndrome or encysted interlobar effusion. For this reason it appeared of interest to report 14 cases of carcinoma situated in the middle lobe bronchus, representing all such cases examined in the Roentgen Department of Sabbatsbergs Sjukhus during the years 1950 to 1954. The aim of the investigation was to establish the distinguishing radiologic features of this condition.

All the 14 cases were operated upon and verified histologically. All patients with one exception were males of ages ranging between 33 and 66 years. The duration of illness varied from 3 months to 1 year with the exception of 2 cases which were detected by chance on mass roentgen examination.

Our material, as regards the routine roentgen appearances, may, in the main, be divided into 3 groups. The first group consists of 3 cases, each of them showing massive infiltration in the right middle lobe and adjacent parenchyma (Figs. 1 and 2). In addition to the primary lesion arising in the middle lobe, secondary deposits involving the adjacent lobes were also demonstrated upon operation in two out of the three cases.

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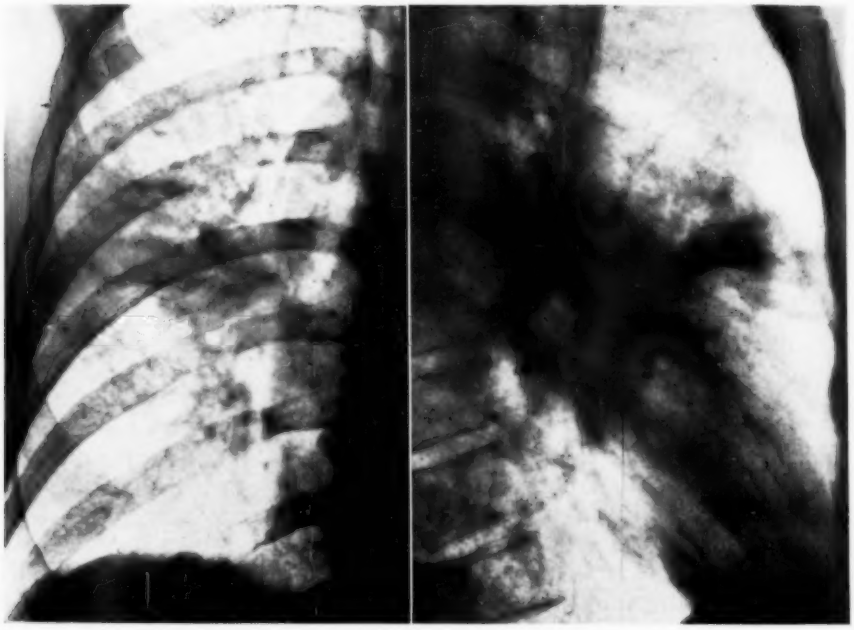

Fig. 1 Fig. 2

Figs. 1 and 2. Case 1. Carcinoma of the middle lobe with secondary deposits in the adjacent parenchyma.

Case 1: Male, aged 60. Duration of illness 6 months. Routine roentgen examination: malignant infiltration of the middle lobe. Bronchography: stenosis of the middle lobe bronchus. Bronchoscopy: tumour of the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 2: Male, aged 66. Duration of illness 1 year. Routine roentgen examination: malignant infiltration of the middle lobe, secondary deposits in the adjacent parenchyma. Bronchoscopy I: inflammation of the mucosa of the middle lobe bronchus. Biopsy specimen: precancerous growth. Bronchoscopy II: stenosis of the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 3: Male, aged 62. Duration of illness 6 months. Routine roentgen examination: malignant infiltration of the middle lobe. Bronchoscopy: stenosis of the middle lobe bronchus. Biopsy specimen: no pathologic change. Bronchography: occlusion of the middle lobe bronchus, the lumen at the site of the occlusion being irregular (Fig. 3). Operation in this case was performed on the basis of the roentgen findings and in spite of the negative biopsy.

These three cases of the first group are, of course, without interest from the point of view of the differential diagnosis.

The second group consists of 6 cases, all characterized by massive infiltration and atelectasis of the middle lobe in the roentgenogram (Figs. 4 and 5). Some years ago similar roentgen appearances were often

Fig. 3. Case 3. Bronchography: irregular transverse occlusion of the middle lobe bronchus (carcinoma).

interpreted as representing encysted interlobar effusions. Bronchography nowadays prevents such misinterpretation. Furthermore, we deny the existence of an encysted interlobar effusion as an independent condition as a matter of principle and consider it as an accessory finding in cases of general exudative pleurisy.

Case 4: Male, aged 61. Duration of illness 3 months. Routine roentgen examination I: malignant infiltration of the middle lobe, first erroneously interpreted as representing an encysted interlobar effusion. Routine roentgen examination II: malignant infiltration of the middle lobe. Bronchoscopy: tumour of the middle lobe bronchus. Biopsy specimen: carcinoma.

Fig. 4 Fig. 5

Figs. 4 and 5. Case 4. Carcinoma of the middle lobe, not to be interpreted as encysted interlobar effusion.

Case 5: Male, aged 65. Duration of illness 3 months. Routine roentgen examination: probably malignant infiltration of the middle lobe. Bronchoscopy: stenosis of the middle lobe bronchus. Biopsy: carcinoma.

Case 6: Male, aged 51. Duration of illness 5 months. Routine roentgen examination: malignant infiltration of the middle lobe. Bronchoscopy: tumour of the middle lobe bronchus. Biopsy specimen: no malignancy. Operation was performed on the basis of the routine roentgenogram in spite of the negative biopsy.

Case 7: Male, aged 66. Duration of illness 7 months. During this period the patient was ill twice with pneumonia situated 'at the base' of the right lung. Routine roentgen examination: infiltration and atelectasis of the middle lobe. Bronchoscopy: no tumour visible in the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 8: Male, aged 48. Duration of illness 4 months. Routine roentgen examination: malignant infiltration of the middle lobe. Bronchoscopy: tumour of the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 9: Female, aged 33. Duration of illness 8 months. Routine roentgen examination: massive infiltration of the middle lobe. Bronchoscopy: stenosis of the middle lobe bronchus. Biopsy: no material could be obtained. Bronchography: occlusion of the

Fig. 6. Case 9. Bronchography; narrow, funnel-shaped occlusion of the middle lobe bronchus (carcinoma).

middle lobe bronchus, the lumen at this site having the shape of a narrow funnel (Fig. 6). Operation was also performed in this case on the roentgen findings and in spite of the negative biopsy.

The third group of patients consists of 5 cases, all having small irregular infiltration and atelectasis of the middle lobe in the routine roentgenograms (Figs. 7 and 8). This group has to be distinguished from cases of middle lobe syndrome.

Case 10: Male, aged 55. Duration of illness 3 months. Routine roentgen examination: irregular infiltration of the middle lobe, probably malignant. Bronchoscopy I: inflammation of the lumen of the middle lobe bronchus. No biopsy was taken. Bronchography:

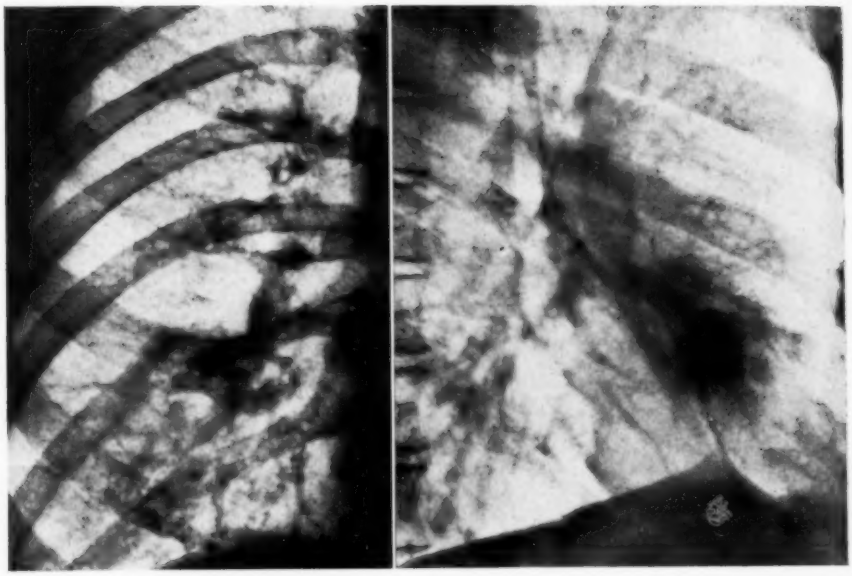

Fig. 7 Fig. 8

Figs. 7 and 8. Case 14. Small, irregular infiltration of the middle lobe (carcinoma).

occlusion of the middle lobe bronchus, although the bronchus lumen was regular in shape at the site of the occlusion. The finding was interpreted as being highly suggestive of a neoplastic lesion although not conclusively pathognomonic (Fig. 9). Bronchoscopy II: occlusion of the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 11: Male, aged 59. Duration of illness 11 months. Two years previously the patient had had pneumonia 'at the base' of the right lung, since when the pneumonic process has not resolved completely. Routine roentgen examination: irregular infiltration of the middle lobe, probably malignant. Bronchoscopy: tumour of the middle lobe bronchus. Biopsy specimen: carcinoma.

Case 12: Male, aged 59. Duration of illness 4 months, the disease having started with cough. One month later, on mass roentgen examination, infiltration in the middle lobe of the lung was detected. Two months later our own routine roentgen examination showed irregular infiltration in the periphery of the middle lobe. The infiltration had increased in size since the mass roentgen examination and a tumour was suspected. Bronchoscopy showed no pathologic findings. Operation was performed on the increasing infiltration in the middle lobe.

Case 13: Male, aged 60. Detected on mass roentgen examination. Routine roentgen examination: irregular infiltration and atelectasis of the middle lobe. Bronchoscopy I: stenosis of the middle lobe bronchus. Biopsy specimen I: chronic bronchitis. Bronchoscopy II: stenosis of the middle lobe bronchus. Biopsy specimen II: possible growth. Bronchog-

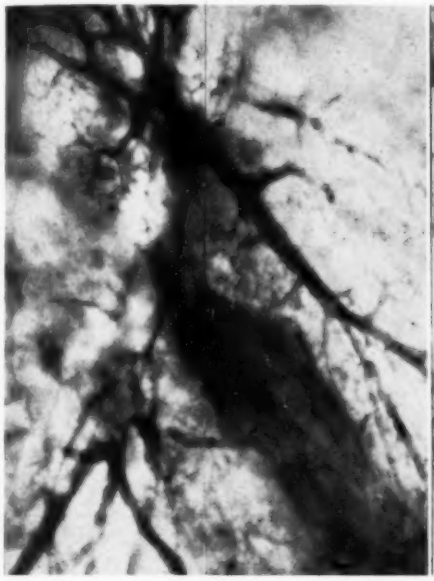

Fig. 9

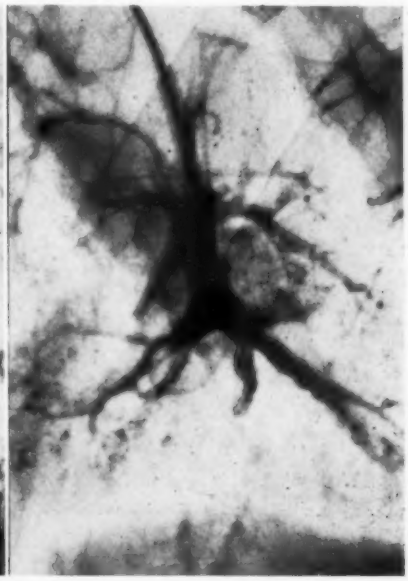

Fig. 10

Fig. 9. Case 10, and Fig. 10. Case 13. Bronchography; occlusion of the middle lobe bronchus, its lumen being smooth and regular at the site of the occlusion (carcinoma).

raphy: occlusion of the middle lobe bronchus, the occluded lumen of the bronchus being smooth — the finding was said to be highly suggestive but not pathognomonic of a growth (Fig. 10).

Case 14 will be discussed on the next page.

Two roentgen methods, namely tomography and bronchography, are available to differentiate carcinoma of the middle lobe bronchus from other conditions in, or in the neighbourhood of, the middle lobe, among which an encysted interlobar effusion and the middle lobe syndrome have particular importance. As to the present investigation, we have no personal experiences of tomography. This method has been reported, however, by ØDEGAARD and by LOCKE as being useful in differentiating the middle lobe syndrome from growths of the middle lobe bronchus.

As regards bronchography, we consider it as being the most valuable roentgen method in differentiating carcinoma of the middle lobe bronchus from other conditions in this region, demonstrating as it does most clearly the shape of the bronchus and the state of its lumen at the site of the occlusion. A number of bronchograms are included to substantiate

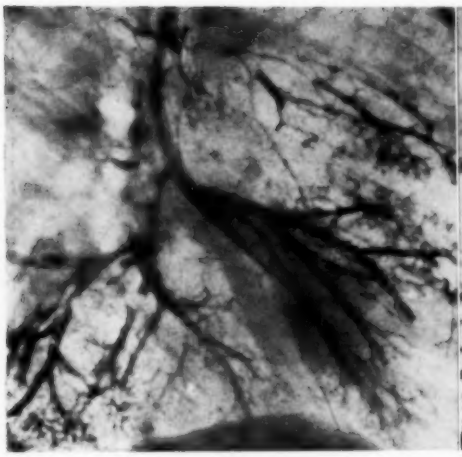

Fig. 11. Case 14. Bronchography; the branches of the middle lobe bronchus are narrow and crowded (carcinoma).

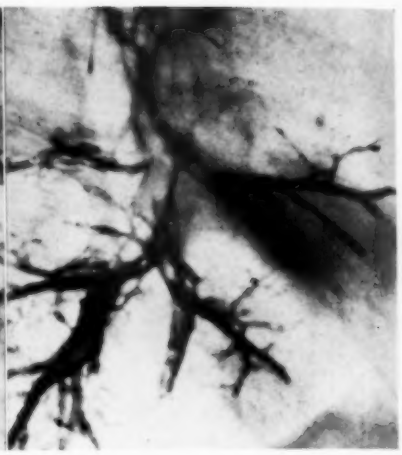

Fig. 12. Same case. Bronchography a few months later. Irregular occlusion of the middle lobe bronchus (carcinoma).

this statement. Figs. 9 and 10 are bronchograms of Cases 10 and 13. The middle lobe bronchus is occluded a few centimetres below the main bronchus in both. The bronchial walls are, however, smooth and regular at the site of the occlusion. Such a finding may be highly suggestive of a growth but cannot be considered as definitely pathognomonic because the filling of the peripheral parts of the bronchus may be prevented by mucus obstructing the lumen. Figs. 11 and 12 are bronchograms performed at various intervals on Case 14.

Case 14: The patient was a male, aged 58, with a previous history of empyema of the right lung at the age of 40. Duration of illness under review 9 months. Routine roentgen examination: irregular infiltration and atelectasis of the right middle lobe of the lung. Bronchoscopy I: no pathologic signs. Bronchography I (Fig. 11): the branches of the middle lobe bronchus are peculiarly narrow and apparently crowded; no peripheral bronchiectasis seen. Presence of a tumour was considered as highly probable, although this was not definitely established by the findings; for this reason further observation of the patient was suggested. Three months later the roentgenograms showed a pronounced increase in the infiltration of the middle lobe. Bronchography II (Fig. 12) performed soon afterwards revealed an abrupt occlusion of the middle lobe bronchus, its lumen being irregular and discontinuous. A few days later the diagnosis of carcinoma was confirmed by bronchoscopy (II) including biopsy.

The bronchograms of Fig. 3 and Fig. 6 have been discussed in detail earlier in this paper in connection with the presentation of Cases 3 and 9.

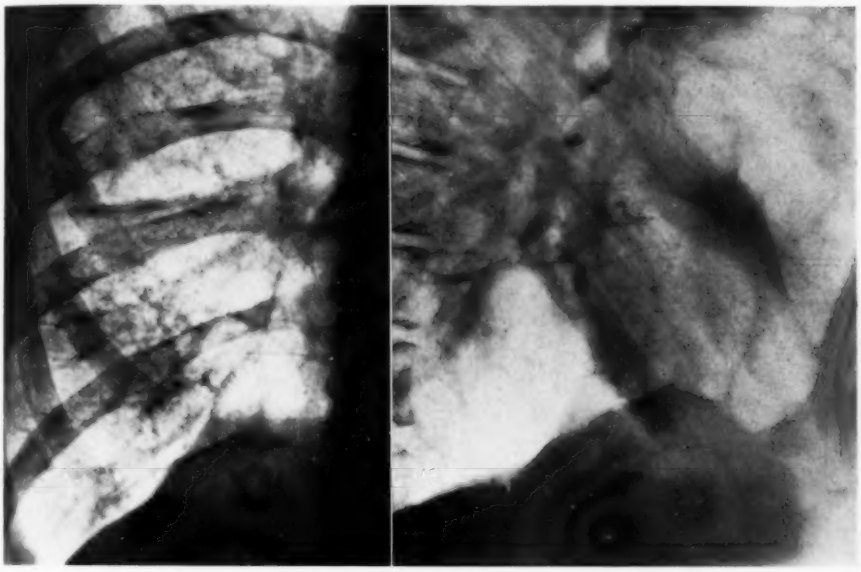

Fig. 13 Fig. 14

Figs. 13 and 14. Middle lobe syndrome. Note the same general features as Figs. 7 and 8.

Both bronchograms reveal characteristic patterns of carcinoma of the middle lobe bronchus, although in different manners.

For the purpose of comparison with the roentgen findings in the middle lobe syndrome, a few suitable roentgenograms are included. Figs. 13 and 14 are the frontal and right lateral views, respectively, of a patient later operated on for middle lobe syndrome. The small irregular infiltrations and the atelectasis of the middle lobe are in general similar to those seen in any of the conventional roentgen films of our cases in Group 3. The bronchogram of Fig. 15, on the other hand, reveals striking differences when compared to the bronchographic findings in our tumour cases. Fig. 15 shows stenosis of the middle lobe bronchus, but its branches are filled behind the stenosis and there are quite large bronchiectatic sacs in the periphery of the bronchial tree. Fig. 16 is a bronchogram of another patient with the middle lobe syndrome. This shows stenosis of the middle lobe bronchus, the stenosis being surrounded by enlarged calcified glands. No peripheral bronchiectasis is demonstrable in this case.

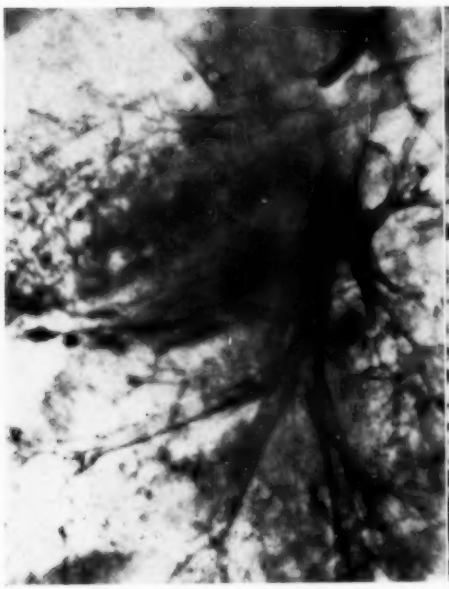

Fig. 15. Bronchography of the same case as Figs. 13 and 14. Local stenosis of the middle lobe bronchus and peripheral bronchiectasis.

Fig. 16. Bronchography: enlarged calcified glands surrounding stenosis of the middle lobe bronchus; no peripheral bronchiectasis seen — proved at operation as middle lobe syndrome.

Conclusions

Persistent infiltrations of any kind in the middle lobe of the right lung are suggestive of the presence of a growth.

To confirm the diagnosis of a tumour, bronchography is considered to be the most satisfactory roentgenologic method.

The diagnosis of a growth of the middle lobe bronchus is established if on the bronchogram,

a) the occlusion of the middle lobe bronchus is irregular and discontinuous,

b) its occlusion is abrupt, transverse, or funnel-shaped.

Bronchograms strongly suggest a tumour of the middle lobe bronchus if they show

a) an occlusion,

b) narrowing and crowding together of its branches.

SUMMARY

A material of 14 operatively verified cases of carcinoma of the middle lobe of the right lung is presented and the differential diagnosis, mainly of the middle lobe syndrome, discussed. The importance of bronchography as a diagnostic method is emphasized.

ZUSAMMENFASSUNG

Ein Material von 14 operativ bestätigten Fällen von Mittellappenkarzinomen der rechten Lunge wird vorgelegt und die Differentialdiagnose, hauptsächlich gegenüber dem Mittellappensyndrom, diskutiert. Die Bedeutung der Bronchographie als diagnostische Methode wird hervorgehoben.

RÉSUMÉ

L'auteur présente un matériel de 14 cas vérifiés opératoirement de cancer du lobe moyen du poumon droit, et discute le diagnostic différentiel, principalement avec le syndrome du lobe moyen. Il souligne l'intérêt de la bronchographie comme méthode de diagnostic.

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THE BEHAVIOUR OF THE PIAL VESSELS DURING AND AFTER THE INTRACAROTID INJECTION OF ROENTGEN CONTRAST MEDIA

by

H. W. Schmidt

Disturbances during cerebral angiography, such as smarting and headache on the side of the injection as well as paralysis and convulsions, are frequently related to vascular injury and cerebral angiospasm, caused by the injection of the contrast media. Many investigators engaged in experimental work on the effect on the vessels of various contrast media have obtained varying results, in themselves somewhat contradictory. In addition to vascular endothelial changes, embolic occlusions and injury to the blood-brain-barrier, as well as angiospasms in various vessel areas, have frequently been reported (4, 5, 6, 7, 8, 9, 10, 17) to be caused by the various contrast media. Other investigators, on the other hand, have observed no vasoconstrictions after injection of such media (1, 16). During angiography of the cerebral vessels in the cat with Perabrodil, marked contraction of the internal carotid artery has occasionally been noticed in our Department; this has only occurred in the extracranial part of the vessel and never within the cranium (14). The behaviour of living pial vessels after the intracarotid injection of roentgen contrast media has been studied by means of microscopic observation and the results are now reported.

Method. Experiments were carried out in rabbits and cats under urethane anaesthesia. After cranial trepanation and careful removal of the dura, the cerebral vessels, constantly irrigated with Ringer's solution, were observed microscopically by means of an Ultropac (Leitz). This method has been fully described in our former publications (12, 13). The various roentgen contrast media (Thorotrast, Perabrodil M 45 % and 80 %, Joduron

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30 % and 70 %, Umbradil 50 %, and Urografin 40 % and 76 %) were injected through a canula, bound into the common carotid artery, in amounts ranging from 1 to 2 ml. The same vessels were observed before, during, and after the injection, and their widths registered by means of an ocular micrometer. In order to gain an objective picture of the width of the vessels and the state of flow, the various stages were recorded photographically (Leica). The vascular widths were measured in the negatives and compared. The blood pressure in the femoral artery was measured during the experiments by means of a mercury manometer.

Results

Observations of blood flow. The contrast medium injected appeared in the pial arteries immediately. When the injection was performed as quickly as possible the vessels for a short time were found to be entirely filled with contrast medium and by reason of the clear watery nature of the contrast medium the vessels were transparent; at the same time a brilliant illumination of the vascular walls was apparent (Fig. 1 b). When the injection was carried out relatively slowly, the vessels were not entirely filled with contrast medium, but a mixture of blood and contrast medium appeared, in which, depending upon the speed of the injection, there was either a preponderance of contrast medium or of blood. Immediately following a rapid injection, the end of flow of contrast medium appeared, followed by the pure blood. A relatively slow injection was followed by a markedly retarded return to normal blood flow with a slowly increasing quantity of blood in the vessels. Shortly after the beginning of the contrast medium had passed through the arteries, it appeared in the veins. The arterial, capillary, and venous phases followed one another succesively, and while they could be observed ocularly, it was not possible for technical reasons to record these various stages photographically. It was only by injecting high percentage solutions, which led to a retardation of the circulation (as will be shown later on in this report), that it was possible to photograph the individual stages successively (Figs. 1 b—e). Even in the case of repeated injections in one animal, the same stage of flow appeared at any one time. On injecting relatively slowly and using a larger quantity of contrast medium, the latter could be detected at the same time both in the arteries and in the veins.

We injected Ringer's solution and Periston (BAYER) = polyvinylpyrrolidon into the carotid artery in controls, whereupon the same conditions of flow were observed as had been seen after the injection of Thorotrast, Joduron 30 %, Perabrodil M 45 %, and Urografin 40 %.

Circulatory disturbances. Whereas no circulatory disturbances were observed after injecting Perabrodil M 45 %, Thorotrast, Joduron 30 %, and Urografin 40 %, there were marked disturbances in the blood flow after injecting high percentage solutions. Thus we found that after the injection of Perabrodil M 80 %, Joduron 70 %, and Urografin 76 %, the

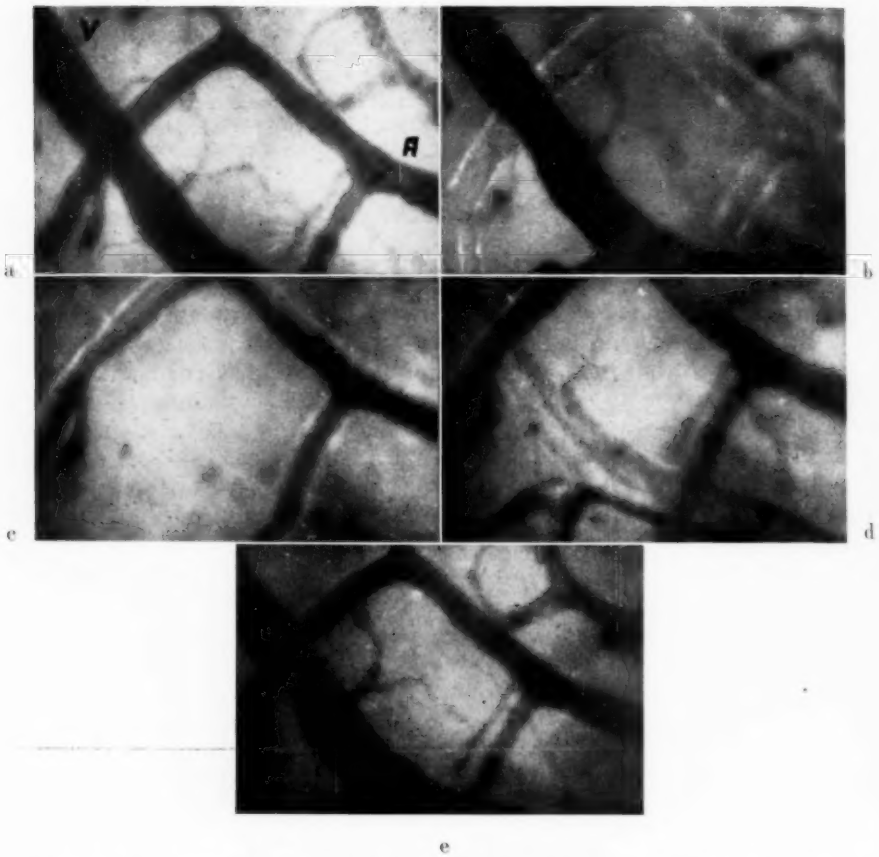

Fig. 1. a. Artery (A) and vein (V) in the pia of a cat before the injection of contrast medium. The diameter of the vessel at the point marked A is approximately 45 μ. b. After injection of 2 ml of Perabrodil M 80 % the arteries are filled with contrast medium. Slight dilatation of the arteries. The veins are still filled with blood (arterial phase). c. The blood flows into the arteries again, whilst now the veins are filled with contrast medium (venous phase). d. The blood begins to flow into the veins again. e. The original condition of flow in the arteries and veins, such as existed before the injection of contrast medium, is restored.

flow in the vessels was retarded and the injected medium often flowed slowly back and forth in the pial arteries. In some cases the circulatory disturbances even led to a temporary arrest of circulation in these vessels. Furthermore, we observed how the lumen of the vessels, when filled with

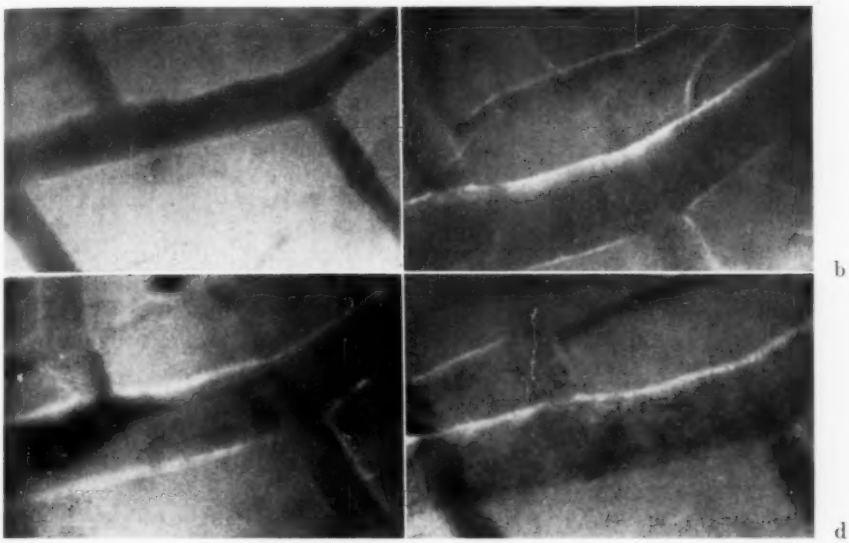

Fig. 2. a. Arteries in the pia of the rabbit before injection of contrast medium. b. The same arteries, dilated after circulatory disturbance caused by the injection of 2 ml of Perabrodil M 80 %. c. The same arteries, dilated after circulatory disturbance caused by the injection of Urografin 76 %. Blood is beginning to flow into the arteries again on the left. d. The same arteries, dilated after circulatory disturbance caused by the injection of 2 ml of a solution of 3 g of Kollidon in 5 ml of Periston.

contrast medium, clearly increased (Figs. 1 b, 2 b and 2 c). When the flow started again, the vessels slowly returned to their original width. The longest period in which these circulatory disturbances occurred was, as observed by us, one minute.

We also made an intracarotid injection of a viscous Periston solution (2 to 4 g of Kollidon of medium molecular weight 9,700 to 5 ml of Periston) in controls. This produced the same circulatory disturbances with vasodilatation as described above (Fig. 2 d). After injecting a solution of 4 g of Kollidon in 5 ml of Periston there was a temporary embolic blocking of the pial arteries; under the microscope we could see a clear, snake-like winding movement of the pial arteries on the surface of the brain, while the animal showed generalized convulsions.

Width of the vessels. We observed the width of the vessels of various diameters (130 to 15 μ). With none of the injected contrast media, not even when highly concentrated, were we able to observe any angiospasm; this applied both when the medium was injected relatively slowly and

at maximum speed, and no changes were observed in the photographs. On injecting the highly concentrated solutions, there was an increase of the lumen of the pial arteries, as described above.

Blood pressure. During and after the injection of the contrast media (with the exception of Thorotrast), the mean arterial pressure measured in the femoral artery, starting from an initial value of 100 mm Hg, fell by 30 mm Hg. This change in blood pressure was gradual, and had mostly reached its lowest point 15 seconds after the start of the injection; after this it slowly rose again. In most cases the previous value had been reached again 40 seconds after the start of the injection. We made an intracarotid injection of the same quantity of Ringer's solution in controls, following which the blood pressure remained unchanged. But after injecting the concentrated Periston-Kollidon solution, the blood pressure likewise fell by 30 mm Hg.

Respiration. We observed respiratory disturbances in a number of cases following the injection of contrast media. In some cases, especially after injecting highly concentrated solutions, acute dyspnea occurred, sometimes followed by apnea which could be overcome by artificial respiration for a short period. Whereas no respiratory disturbances were observed after injecting Ringer's solution and Periston, such disturbances occurred after injecting a concentrated Periston-Kollidon solution.

Convulsions. In isolated cases, especially after Joduron, generalized convulsions occurred during or immediately after the injection of highly concentrated solutions of contrast media. Whereas after injecting Ringer's solution and Periston, no such convulsions were observed, they were found to be particularly marked after injecting a concentrated Periston-Kollidon solution.

Discussion

The method of observing the living pial vessels enables us to study the conditions of flow and the behaviour of the vessels in cerebral angiography. The successive arterial, capillary, and venous phases, may be clearly followed under the microscope. It is found that when the contrast media are injected rapidly in animal experiments, a condition is achieved in which the entire lumen of the vessels is filled with contrast medium, whereas when the injection is effected more slowly, a mixture of blood and contrast medium flows through the vessels. In the case of cerebral angiography in man, complete filling of the vessels with contrast medium will hardly be possible, as the injection is performed with a relatively small canula into the vessel (either the common carotid or the internal carotid artery). The contrast medium immediately mixes with the blood flowing from the heart through the great vessel, so that it is always a mixture of

contrast medium and blood which flows through the vessels. Animal experiments, however, confirm the clinical experience that injection at maximum rapidity produces the highest possible concentration of contrast medium in the cerebral vessels.

It seems to be of some importance that we could show by our method that after an intracarotid injection of Perabrodil M 45 % and 80 %, Joduron 30 % and 70 %, Thorotrast, Umbradil 50 %, and Urografin 40 % and 76 %, no angiospasm of the intact pial vessels occurred. We realize that these findings are contrary to those of many authors, and for this reason, as well as on account of the importance of the spasm problem, we should like to consider this problem more closely. Our result corresponds with the observations of BASSETT, ROGERS, CHERRY and GRUZHIT (1) who observed no acute spasm of the pial vessels after injecting Diodrast.

FOLTZ, THOMAS and WARD (8) found that in monkeys in which they observed the pial vessels while measuring the intracranial pressure, Diodrast produced brief, violent spasms of the cerebral arterioles which started in the middle of the injection. "This began as a high speed, repetitive series of momentary arteriolar constrictions which gave a fluttering appearance lasting only a few seconds. This was replaced by a cortical blanching in which the dark veins stood out in startling contrast. This second phase always began during the end of injection." This was followed by a third phase "consisting of a cortical blush lasting 1 to 2 minutes".

It is doubtful to us whether it may be concluded from these observations that vasoconstriction or dilatation of the pial arterioles takes place. The picture presented is quite in accordance with the circulatory disturbances which we saw under the microscope. The cortical blanching would correspond to the circulatory arrest in the case of the dilated pial vessels when filled with the contrast medium and the subsequent reddening to the stage in which the dilated vessels fill up with blood again and which, as we have described above, slowly return to their original lumen. Similarly, the change in intracranial pressure observed by FOLTZ et coll. is in accordance with the circulatory disturbances without angiospasm which we observed.

If BROMAN and OLSSON (4) attribute their finding — that, after the intra-arterial injection of Diodrast, the blood is not visible in the pial vessels for 3 to 8 seconds besides lowering of the blood pressure — to an angiospasm, then, in single cases, angiospasm of the extracranial vessels may also be the cause, as has been observed in other tests at this Institute. This angiospasm, however, is by no means a regular occurrence. According to our findings, the retarded blood flow in these cases is caused by hypotension with simultaneously increased viscosity.

We cannot agree with HOLM (9) when he concludes that because, in the cerebral angiogram, the vessels were somewhat narrower after Perabrodil than after Thorotrast, the former produces a vascular contraction; this difference in the angiogram may also be caused by other factors. WEATHERALL (17), for example, gives an illustration showing a roentgenogram of tubes of equal calibre filled with various contrast media which not only differ in intensity but also in shadow width.

The observation in various experimental works (7, 17) that vasoconstriction of the vessels in the limbs with subsequent dilatation following the injection of roentgen contrast medium occur, is not in absolute opposition to our results, since it has been repeatedly pointed out that the cerebral vessels react differently from those in the limbs. It would therefore appear quite possible that roentgen contrast media in the cerebral vessels do not lead to angiospasm, whereas they might cause a spasm in the arteries of the limbs. As in our findings with the pial arteries, STÖR (16), too, found no change in the blood flow in the mesenteric arteries after the intraarterial injection of Uroselectan, Abrodil, and Thorotrast, but sometimes a slight dilatation.

The circulatory disturbances in the pial vessels following the injection of highly concentrated contrast media is, in our opinion, of particular importance. We have already stressed the fact that we did not observe such circulatory disturbances after using Perabrodil M 45 %, Joduron 30 %, Urografin 40 %, and Thorotrast. As no circulatory disturbances occurred after the injection of Ringer's solution and Periston in control tests, but, on the other hand, these were observed quite definitely after using Periston-Kollidon solutions, accompanied by convulsions and respiratory disturbances, we are inclined to attribute the circulatory disturbances described above, which occurred following the injection of concentrated contrast media, to other physical properties, possibly to viscosity. Stasis first occurs in the smaller vessels and then continues backwards into the larger arteries, resulting in a circulatory arrest and, consequently, as a result of lack of oxygen, to dilatation of the vessels. This can be produced particularly clearly by the injection of a contrast medium under pressure. We believe that the circulatory disturbances we have described may be regarded as the actual cause of the complications occurring in cerebral angiography, such as smarting and headache on the side of the injection, paralysis, and convulsions. The damage to the vascular walls with permeability disturbances, hemorrhage, edema, and changes in the neuroglia and ganglion cells, such as have been described after cerebral angiography (2, 4, 5, 6, 10, 16), would then be a consequence of these circulatory disturbances of longer duration.

The fact that functional circulatory disturbances in the brain may

produce similar parenchymal changes as a vascular arrest by embolism or thrombosis, has already been shown by BODECHTEL and WICHMANN (3). We regard the findings of BROMAN, FORSSMAN and OLSSON (6) as further confirmation of our view. They were able to show that after artificial stasis produced by occlusion of the afferent brain vessels, greater injury to the cells was produced by the contrast medium than was the case without such occlusion.

Our findings are also in accordance with the investigations carried out by BLOOR, WRENN and MARGOLIS (2) who, in histologic tests, observed a general dilatation of the parenchymal vessels of all calibre as an early change after cerebral angiography and, only later, hemorrhage and cellular changes. Our observations on the change in blood pressure after the injection of a contrast medium into the carotid artery and on the associated respiratory disturbances and convulsions correspond with the findings of numerous authors (4, 5, 6, 10, 11, 15, 17).

SUMMARY

After intracarotid injection of various contrast media on rabbits and cats under urethane anaesthesia no angiospasms of the intact pial vessels were observed. On the other hand, circulatory disturbances were observed after injecting concentrated contrast media. Such disturbances were also found after injecting concentrated Kollidon-Periston-solution. These circulatory disturbances are therefore attributed to other physical properties (possibly viscosity) with simultaneous decrease of blood pressure and they are regarded as the real cause of the various complications occurring in cerebral angiography.

ZUSAMMENFASSUNG

Bei Kaninchen und Katzen in Urethannarkose wurden nach Injektion verschiedener Röntgen-Kontrastmittel durch die A. carotis keine Angiospasmen der intakten Pia-Gefässe beobachtet. Nach Injektion von konzentrierten Kontrastmitteln werden Kreislaufstörungen beschrieben, wie sie nach Injektion von konzentrierter Kollidon-Periston-Lösung zu beobachten sind. Diese Kreislaufstörungen werden daher auf andere physikalische Eigenschaften (möglicherweise Viskosität) bei gleichzeitiger Blutdruckerniedrigung zurückgeführt.Diese Kreislaufstörungen werden als eigentliche Ursache für verschiedene bei der cerebralen Angiographie auftretende Zwischenfälle angesehen.

RÉSUMÉ

L'injection intracarotidienne expérimentale de différents produits de contraste chez des lapins et des chats sous anesthésie à l'uréthane n'a pas permis d'observer de spasme des vaisseaux pie-mériens intacts. Mais des troubles circulatoires ont été observés après l'injection de produits de contraste concentrés. L'injection d'une solution concentrée de Kollidon-Periston a aussi donné lieu à des troubles identiques. C'est pourquoi ces troubles circulatoires sont attribués à d'autres propriétés physiques (probablement viscosité), lors d'une diminution simultanée de la pression sanguine, et sont considérés comme la cause réelle des diverses complications survenant au cours de l'angiographie cérébrale.

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FROM THE DEPARTMENT OF MEDICINE (DIRECTOR: PROFESSOR F. S. P. VAN BUCHEM), THE PATHOLOGY LABORATORY (DIRECTOR: PROFESSOR J. J. TH. VOS), AND THE ROENTGEN INSTITUTE (DIRECTOR: PROFESSOR S. KEYSER), OF THE UNIVERSITY CLINIC, GRONINGEN, THE NETHERLANDS

AN UNCOMMON FAMILIAL SYSTEMIC DISEASE OF THE SKELETON: HYPEROSTOSIS CORTICALIS GENERALISATA FAMILIARIS

by

F. S. P. van Buchem, H. N. Hadders and *R. Ubbens*

This paper reports cases of a twin brother and sister with a peculiar systemic disease of the skeleton, which notwithstanding accurate clinical observation over a number of years and, in one of the cases, extensive post-mortem examination, has not fitted into any classification of diseases of the skeleton so far described. It is true that signs which also occur in known bone diseases were found, although in these there was also a considerable number of dissimilarities.

Case 1. Male, aged 48 years, was admitted with meningococcal meningitis; recovery following treatment with penicillin. He had attended school till his eleventh year and remained two years in one class. Worked as a farm labourer up to his 32nd year, since when, unable to continue his occupation, he became a hawker. He regularly went out on a bicycle with a fairly large load on his back up to half a year previously. According to photographs, he had a normal appearance when 10 years of age but, at the age of 25, his chin was already large and broad, suggesting acromegalia. At the age of 37, he was no longer able to see well. An examination showed papilloedema with atrophy of the optic nerves, as well as hyperostosis of the skull bones. At that time he tired rapidly and since then had had occasional vertigo. During the last few years walking, as well as speech, became difficult. Gradually he became more deaf, and was now and then troubled by stabbing head pains. Health otherwise good.

Clinical examination: Well-built man; good nutritional condition. The skin was dry and slightly flaky but not thickened or hardened. Height: 1.80 m, weight: 64 kg. Conspicuously high forehead and large prominent chin, caused by the greatly thickened lower jaw, not painful to pressure. The teeth were ground off and in a normal position in the lower jaw. Normal bite. In contrast to acromegalia there was no prognathism and the teeth occupied normal space in the mandible. Mouth-opening rather small; tongue in normal size. Slight exophthalmus with a wide eye-fissure. The pupillae were equally round, reacted well to light and convergence. Discs: atrophy of the optic nerves on both

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sides; vision on both sides 1/36, not correctable; fields of vision could not be determined. The thorax was well arched, did not expand on respiration, and was not painful to pressure. The clavicles were very thick and broad. Extremities: poorly developed muscles; the bones thickened, not painful to pressure. Vertebral column rigid. Thyroid, genitalia, heart, lungs and hair normal.

Laboratory examination: Urine: N. A. D. Blood: N. A. D. Blood calcium content 9.9 mg %; inorganic phosphate 2.67 mg %; acid phospatase 1.5 U; alkaline phosphatase 19 U King Armstrong: cholesterol 1.49 ‰. Syphilis tests: negative. Blood sugar curve following 50 g glucose: fasting 115 mg %, 163 mg % after ½ hr, 212 mg % after 1 hr, 148 mg % after 1½ hrs, 98 mg % after 2 hrs, 90 mg % after 2½ hrs; no glucose in the urine. Adrenaline test: eosin 10 × 3; 0.3 mg adrenaline: 7 × 3 after 3 hrs, 8 × 3 after 4 hrs, 7 × 3 after 5 hrs.

Table 1

Thorn test:

eosin. leuc.	eosin.	bas.	stabs.	polyn.	lymph.	mon.	uric acid creatinine ratio
72 × 3 9,500	6 %	—	—	59 %	33 %	2 %	0.61/1.01 = 0.6
25 mg ACTH:							
after 3 hrs:							
45×3	3 %	—	1 %	82 %	12 %	2 %	0.60/0.98 = 0.61
after 4 hrs:							
19 × 3 10,700	1 %	1 %	1 %	86 %	9 %	2 %	
after 5 hrs:							
12 × 3 9,600	—	1 %	2 %	89 %	7 %	1 %	0.81/1.15 = 0.70

Fig. 1. Case 1. Thickened and sclerotic bone structure in the clavicle, ribs and humerus.

Insulin test: blood sugar content fasting 117 %, 6 U insulin intravenously: 74 mg % after 20 min., 105 mg % after 40 min., 100 mg % after 60 min., 106 mg % after 90 min., 110 mg % after 120 min.

The hormone assay of the urine (E. DINGEMANSE) showed the following result: oestrogen substances 50 I. U. per 24 hrs, gonadotrophins less than 40 U. total 17-ketosteroids 8.1 mg per 24 hrs.

After a diet containing 1 g calcium per diem, in three days 364 mg was excreted in the urine, in the faeces 3,877 g, totalling 1.4 g per day, of which 121 mg was in the urine.

No neurologic disorders were found. In the meantime, the patient had, however, become feeble minded. Figs. 1 to 3 and 8 E show the result of the roentgen examination.

Fig. 2. Case 1. Thickened and sclerotic skull-cap. Enlarged and sclerotic mandible.

Case 2. The twin sister, aged 52, of the above-mentioned patient, had always been mentally deficient, but was able to do her simple duties as a farm maid up to the age of 20. At 10 years of age her appearance was normal, but at the age of 20 she had a large chin with slightly protruding eyes. At that time she could see and hear well, and menstruation was regular. Since then she gradually became deaf and the power of vision decreased; from the age of 40 completely blind and unable to hear anything. She gradually gained in body weight. When 28 years old she showed marked papilloedema; the fields of vision were intact. At that time hyperostosis of the skull bones and the majority of the long bones was found; otherwise she had allegedly never been ill. Her father died when 66 from heart disease, her mother from diabetes at the age of 92. *The parents were not related.* No other members of the family had disorders of the jaws or eyes.

Clinical examination at the age of 50: Considerable amount of fat, especially on the shoulders and upper arms. The patient occasionally ejaculated inarticulate sounds. Skin not hardened or thickened, but soft and glossy with striae on the abdomen and on the shoulders. Axillary hair-growth absent, pubic hair-growth very scanty. The head showed the same abnormalities as in her brother, viz. high forehead, prominent chin, caused by a broad and greatly thickened lower jaw, not painful on pressure, no prognathism, good bite. Mouth-opening rather small, the tongue normal in size. The eyes protruded slightly, eye-fissure wide. The pupillae were equal in size, round, and did not react to light. Discs: atrophic papillae and tortuous vessels on both sides. The distance between the outer

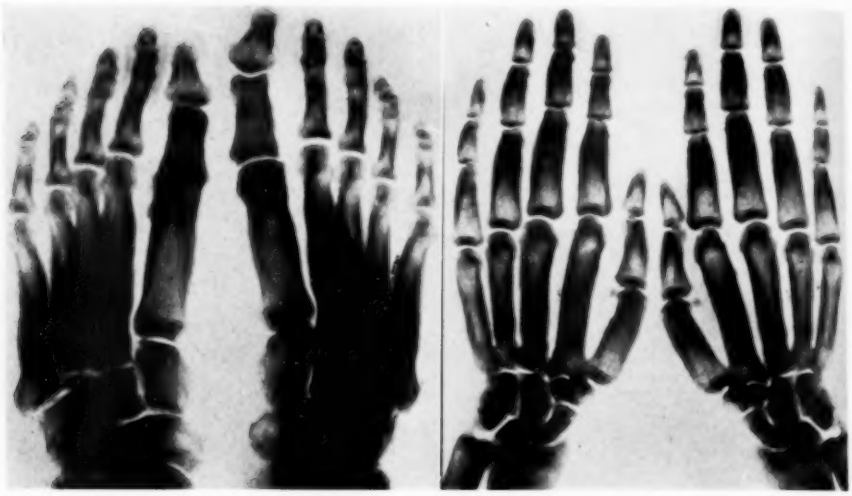

Fig. 3. Case 1. Thickened metacarpal and metatarsal bones with periosteal crests.

edges of the orbits was 12 cm. Thorax well-arched, the clavicles thickened, the thorax showed little movement on respiration. Vertebral column rigid. Flexion of the neck was difficult but rotation of the head possible. As regards the extremities, the slight muscular development was noticeable, the muscles hypertonic. The tendon reflexes were exaggerated, no pathologic reflexes. Joints not swollen. Movement of the knees good, of the hips difficult. Heart, lungs and thyroid not abnormal.

Laboratory examination: Urine: N. A. D. Blood: N. A. D. Blood calcium content 9.3 mg %, inorganic phosphate 2 mg %, acid phosphatase 2 U, alkaline phosphatase 21 U King-Armstrong. Syphilis tests: negative. Total protein: 5.47 %, albumin 3.26 %, globulin 2.21 %. Blood sugar curve, 50 g glucose, fasting 102 mg %, 139 mg % after $^1/_2$ hr, 134 mg% after 1 hr, 135 mg% after $1^1/_2$ hrs, 112 mg% after 2 hrs, 97 mg% after $2^1/_2$ hrs.

Table 2

Adrenaline test:	eosin.	leuc.	eosin.	bas.	stabs.	polyn.	lymph.	monoc.
	162 × 3	11,800	7 %	1 %	1 %	55 %	30 %	6 %
0.3 mg adrenaline:								
after 3 hrs:	85 × 3	8,300	6 %	—	1 %	55 %	34 %	4 %
after 4 hrs:	53 × 3	8,600	3 %	—	2 %	66 %	25 %	4 %
after 5 hrs:	52 × 3	9,500	3 %	1 %	1 %	67 %	26 %	2 %

Insulin test, 6 U insulin intravenously: blood sugar fasting 108 mg %, 100 mg % after 10 min., 102 mg % after 20 min., 90 mg % after 30 min., 80 mg % after 40 min., 90 mg % after 60 min., 56 mg % after 90 min., 68 mg % after 120 min.

The hormone assay of the urine (E. DINGEMANSE) showed the following result: total 17-ketosteroids 4 mg per 24 hrs.

Figs. 4 to 7 and 8 E show the result of the roentgen examination. Two years later the patient developed broncho-pneumonia and died.

Summary of autopsy findings. The skeleton showed conspicuous changes. The skull had a greatly thickened cap and base (Fig. 5); the facial bones had increased less in size although the lower jaw was considerably enlarged (Fig. 6). The weight of the skull was 2,357 g, that of the lower jaw 265 g. The thickened bone tissue in the plane of section was very compact, the surface presenting excrescences. These, mostly small, excrescences were plateau-shaped or tapering off to a point, like osteophytes. Similar excrescences were found in all other parts of the skeleton in varying numbers; many arose from the ribs and many of the long bones (Fig. 7), fewer from the remaining long bones and from the short bones, and a few from the flat bones. In the plane of section the corticalis of the long and short bones appeared considerably thickened in the diaphyses but not in the epiphysis (Fig. 8 E); hence the spongiosa was smaller but otherwise unchanged. The other parts of the skeleton likewise showed an unchanged spongiosa structure. Histologic examination revealed that a number of rounded bony lamellae had been deposited subperiosteally; there were small osteophytes locally. The chemical composition of the bone tissue remained within normal values. Osteoblasts and osteoclasts appeared sporadically; consequently there was no marked production or destruction of bone. The bone marrow was macroscopically and microscopically without conspicuous changes. The light weight, 930 g, of the brain was a striking feature. The pituitary gland had changed into a vesicle with a very thin-walled structure, without histologic remnants of the adenohypophysis. The parathyroid glands were not found; serial sections were, however, not made. An obturating thrombus was situated in the lower vena cava. The lower lobe of the right lung showed bronchiectases and bronchopneumonic foci; an obturating thrombus mass was found in a large arterial branch of this lobe.

Fig. 4. Case 2. Thickened and sclerotic corticalis of tibia and fibula with irregular periosteal crests.

Discussion

There are a few diseases which must be taken into consideration in establishing a diagnosis in these cases.

1. *Osteosclerosis* (*osteopetrosis*) *congenitalis diffusa* or *marble-bone disease of Albers-Schönberg.* This is a hereditary disease in which the heredi-

a

b

Fig. 5 a and b Case 2. Exterior of skull post mortem.

ty may be dominant or recessive (Cocchi). There is thickening of the corticalis especially in the epiphyses and metaphyses of the long bones (Fig. 8 B→C), and of the spongiosa of all bones. The bone marrow diminishes in extent and may be even completely obliterated. There is formation of fresh bone tissue, but no destruction of the tissue already formed. The osteosclerosis principally affects the vertebrae, the pelvis, and the metaphyses and epiphyses of the long bones.

It is precisely the vertebrae and the pelvis that are much less affected in our cases; in the long bones the diaphyses were mostly involved. Furthermore, we found gross changes in the skull and ribs, which are mostly unaffected in Albers-Schönberg's disease. By the reduction of the marrow space, the liver and spleen are generally enlarged by extra-osseal haematopoesis, so that normoblasts and myelocytes occur in the peripheral blood (leuco-erythroblastic blood picture). This was not evident in our cases.

2. *Osteomyelosclerosis.* In this disease the endosteal sclerosis is prominent, due to an increase in the number and size of the osseous bars in all parts of the skeleton, especially those of the trunk. Increase in width of the cortex (of the diaphysis) is also possible, the shape of the bones as a rule remaining, however, unchanged. The marrow cavity becomes greatly

Fig. 6. Case 2. Interior of skull post mortem.

reduced; furthermore, the marrow is noticeably lacking in cells; it consists of fibrous connective tissue with only an occasional cellular focus, so that in this disease an extra-osseal haematopoesis also occurs. Our cases, as mentioned before, exhibited no signs of haematopoesis outside the skeleton.

3. *Hyperostosis generalisata, with pachydermia* (UEHLINGER). This disease starts in puberty with pain in the joints, especially of the feet, and rapid tiring. The joints of the feet, hands, and knees, become swollen. The small joints of the feet and hands become rigid due to synostosis.

The build of the body is disproportionate: the extremities are relatively long, with the features suggesting acromegalia. Pachydermia is present in the forearms and legs, the skin of the forehead and the scalp being sometimes also affected. Clubbing of the fingers and toes may occur. The disease is of a recessively hereditary character.

In the long bones (Fig. 8 C), the corticalis is sclerotic in its entire length, including the region of the epiphysis. The ribs, collar-bones and pelvis are also sclerotic. New bone is deposited sub-periosteally, mostly in the form of spongy bone, and osteophytes are also present. The originally compact corticalis is broken down and replaced by more or less cancellous bone tissue. The skeleton is increased in size. In the spongiosa

Fig. 7. Case 2. Tibia post mortem.

itself, a great number of osseous bars disappear, while the remainder are strikingly heavy and arranged by mechanical influences; this process is called sclerosing atrophy. The marrow between these thickened osseous bars consists of fatty marrow with only a small quantity of normal cellular marrow; there is no fibrosis. The skull shows no periosteal bone deposition, but only endosteal hyperostosis of petrous bone. Ossification of the small joints of the vertebrae and ribs as well as of the joints of the hands and feet occurs. As a result of pressure on the nerves, radical pain, pareses, sphincter palsies, and such, may arise.

In our cases no sclerosing atrophy was found in the bone marrow, whereas the thickened corticalis indeed consisted of very compact bone tissue, but showed a normal width in the region of the epiphysis (Fig. 8 E). The changes of the skull were most striking. Synostosis of the small joints of the hands and feet was absent; there was, however, synostosis of the vertebral column. In one of the cases, the hypophysis had become a vesicle.

Hyperostosis generalisata must be distinguished from osteo-(arthro) pathia hypertrophicans toxica ('ostéoarthropathie hypertrophiante pneumique Marie-Bamberger'). This latter is an acquired disease, in which fresh bone tissue is subperiosteally deposited radially and tangentially on an unchanged corticalis. This cuff-shaped bone increase in the extremities affects the long bones particularly and the short bones less. The skeleton of the trunk is rarely affected.

4. *Osteopathia hyperostotica multiplex infantilis or Camurati-Engelmann's disease.* In this disease, as in our cases, the symmetric osteosclerosis is localized more to the diaphyses of the long bones and phalanges of the

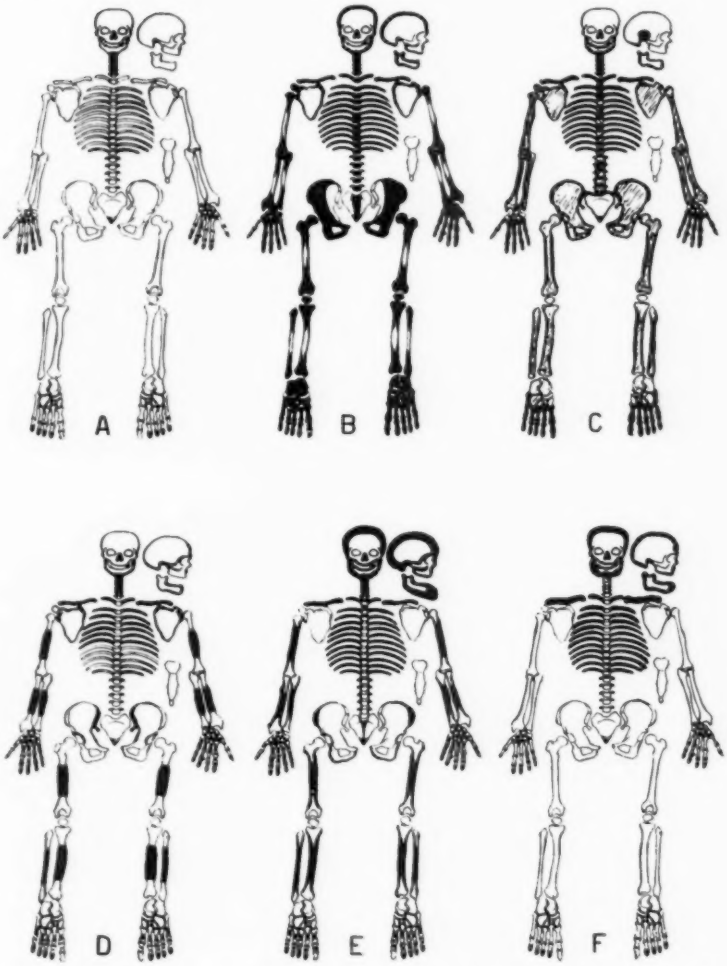

Fig. 8. Diagram of the skeleton. A. Normal. B. Marble bones, Albers-Schönberg's disease. C. Hyperostosis generalisata. D. Camurati-Engelmann's disease. E. Authors' case. F. Halliday's case.

hands and feet which are, however, spindle-like, thickened, and with a rather sharp boundary between them and the metaphyses and epiphyses, which remain free (Fig. 8 D). The vertebrae and the pelvis are rarely affected and the skull less extensively. Periosteal deposition of bone tissue is present and the compacta may be broken down from within.

The bone marrow becomes fibrotic. This is likewise a hereditary disease, starting in infancy. The patients have pain in the legs and are unable to walk properly (NEUHAUSER). They tire rapidly and the muscles and fatty tissue show atrophy. A slight anaemia is sometimes found, but otherwise there are no blood changes. The arms and legs are relatively long. RIBBING described a hereditary form of osteosclerosis with the same localization in the diaphyses. As points of difference, he mentioned its occurrence at a slightly older age, viz., during and after puberty, and the finding that the disorders remained restricted to the long bones of the lower extremities.

COCCHI gave a picture of an older patient (37 years), whom he considered to be suffering also from Camurati-Engelmann's disease. STODTMEISTER and SANDKÜHLER count this disease of Camurati-Engelmann among the hyperostosis generalisata, which is distinguished by its onset at an earlier age.

The main differences in our cases therefore are: the much less marked skull changes, the spindle-like thickening of the diaphysis of the long bones, the fibrosis of the marrow, and the onset at an early age (Fig. 8 D and E).

When searching the literature we found 3 descriptions of skeletal disorders which in part show much similarity to those of our cases.

As far as the skull is concerned, our cases in the first place resemble a patient, described by JOURDAIN in 1778, later by LEBERT in 1857, and finally mentioned by VIRCHOW. It concerned the son of a surgeon who, at the age of 12 (1734), began to show a slow-growing swelling of the right maxilla. In three years this caused complete occlusion of the nose, so that respiration was only possible through the mouth. Enlargement of the other facial bones and the lower jaw together with exophthalmus produced a monstrous appearance in the patient when he was 20. He became blind and died at the age of 45. LEBERT depicted beautiful drawings of the greatly thickened skull (Fig. 9), the weight of which was 4,250 g. The lower jaw alone weighed more than 1,500 g. It was noteworthy that the remainder of the skeleton presented strikingly thin bones.

Similar marked skull abnormalities were shown in the patient described by HALLIDAY. This was a girl, 10 years old, with changes in the skull, the lower jaw, the collar-bones, and the ribs (Fig. 8 F). The sclerosis of the collar-bones and the ribs, however, was localized mainly to the central portions. This girl also had atrophy of the optic nerves and was deaf (nerve deafness). The difference in our cases consisted in finding the changes along the entire length of the long bones; on the other hand, the cortices were thin, only the 1st metacarpal bone and the proximal part of the radius showing sclerosis. Then again, the other changes in the skeleton in our cases in many respects correspond to those described exten-

sively by ARNOLD. This concerned a man of 48 years in whom the bones of hands, feet and legs gradually increased in size about 30 years previous to his death. At autopsy the skull appeared almost unchanged. On the other hand, nearly all other bones of the skeleton were covered with osteophytes. The corticalis was thickened and the marrow cavity smaller than normal. Microscopically, the increase in volume appeared mainly to be caused by subperiosteally deposited bone tissue, especially in the diaphyses. In addition, osteophytes had been formed in every situation. There was barely any trace of active bone production.

Fig. 9. Skull of Lebert's patient.

A striking difference in our cases was the absence of skull abnormalities, as well as the fact that ARNOLD's patient showed signs of pachydermia. A brother showed the same disorders clinically. ARNOLD's patient was probably a case of hyperostosis generalisata with pachydermia. We therefore come to the conclusion that the clinical and morphologic picture of our cases cannot be classified among any of the known syndromes.

The abnormalities of the bones consist mainly of marked hyperplasia of the corticalis with extensive osteophyte formation; the size of the bones was increased and the marrow cavity reduced. The thickening of the corticalis was chiefly localized to the diaphysis of the long bones, the metacarpals, metatarsals and phalanges. The spongy bone showed an ordinary structure. There were no abnormalities of the internal organs. The calcium and inorganic phosphate content of the serum were normal and the alkaline phosphate slightly increased. The blood picture was normal. Although at autopsy the pituitary gland appeared reduced to a vescicle in the wall of which no adenohypophysial elements could be found microscopically, viz., only glia tissue and a few mononuclear lymphocyte-like cells, the other endocrine glands were normal; and endocrine disorders were also absent clinically.

The condition occurred in a twin brother and sister. The remainder of the family was healthy. Hence the cause might have arisen from germinal damage during pregnancy.

Our cases, in some respects, show resemblance to those of Camurati-Engelmann's disease.

SUMMARY

Two cases presenting uncharacteristic systemic bone changes occurring in a twin brother and sister are described. The differential diagnosis is discussed in detail and certain resemblance of the cases to those of Camurati-Engelmann's disease pointed out.

ZUSAMMENFASSUNG

Zwei Fälle mit uncharakteristischen Systemveränderungen des Skelettes bei Zwillingen, einem Mann und seiner Schwester, werden beschrieben. Die Differentialdiagnose wird eingehend diskutiert und eine gewisse Ähnlichkeit der Fälle mit denen der Camurati-Engelmann'schen Krankheit wird hervorgehoben.

RÉSUMÉ

Description de deux cas de modifications du système osseux non caractéristiques chez un frère et une soeur jumeaux. Le diagnostic différentiel est discuté en détail et certaines ressemblances de ces cas avec ceux de la maladie de Camurati-Engelmann sont mises en évidence.

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KINKING OF THE AORTA

Report of two cases

by

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An uncommon abnormality of the aorta in the region of the junction of the aortic arch and the descending aorta has been recently reported. Just distal to the origin of the subclavian artery, the aorta, instead of curving upwards and backwards as usually, swerves forwards and downwards as far as a point near the attachment of the ligamentum arteriosum Botalli. The vessel then kinks abruptly backwards and laterally until it resumes its usual course. The appearance resulting from the deformity closely resembles that which is commonly encountered in coarctation of the aorta, although no clinical or roentgenologic evidence of stenosis of the vessel is to be found.

This condition is of interest, particularly from a roentgenologic angle, inasmuch as it may be associated with marked mediastinal deformation and likely to lead to an erroneous diagnosis.

Three cases of this anomaly were first reported by SOUDERS, PEARSON and ADAMS in 1951. In the first of those cases, a routine examination of the chest had revealed the presence of a clearly outlined, rounded opacity of soft-tissue density, measuring 5.5 mm in diameter, overlying the aortic arch but somewhat more to the left of the midline.

A tentative diagnosis of Hodgkin's disease with mediastinal involvement had been made, and a course of irradiation therapy instituted. However, no modification of the roentgenologic appearance was observed to follow this treatment, so that surgery was finally resorted to. At operation, the aorta was found to present the abnormality and elongation described above, while a slight patency of the ductus arteriosus was also detected.

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As regards the other two of the cases reported, conventional chest films disclosed roentgenologic appearances that were similar to the one just mentioned; these findings were further substantiated by angiocardiography.

After pointing out that no such observations had previously been made except for a case briefly outlined by REICH, the above workers continued: "We wish to suggest that there is yet an additional lesion which so far as we are aware has not previously received attention in the literature. This is the double curving or buckling of the aorta, which in the postero-anterior projection of the roentgenogram gives the appearance of a soft tissue mass superior to and overlying a small or normal aortic arch."

The observation of two more cases has since been reported by DOTTER and STEINBERG, in which angiocardiography revealed an aortic deformity closely resembling the one usually associated with coarctation, although no evidence of stenosis of the vessel could be detected.

To these six we are in a position to add two more cases which we had the opportunity to observe.

Case 1. V. H., aged 27, had, since early childhood, suffered from marked weakness, especially pronounced early in the day. On being admitted to hospital at the age of 25 for treatment of a tonsillar abscess, 'congenital heart disease' was suspected. Subsequently, the patient complained of attacks of praecordial palpitation, slight dyspnoea on exertion, and vague chest pain. In October 1951, he was sent to Södersjukhuset for investigation.

Physical examination: Good general condition. A harsh systolic murmur of short duration was heard, its point of maximum intensity being over the apex. There was no cyanosis, oedema, or dyspnoea.

Roentgen findings: The cardiac silhouette showed normal pulsation. Total volume was 700 cc, corresponding to 300 cc per square meter of body surface. There was neither enlargement nor deformity of the heart. On the left, a semispherical opacity of soft tissue density, having an even, sharply defined contour, overlying the aortic arch, and projecting beyond the left border of the mediastinum, was detected (Fig. 1). Another rounded shadow of greater density was superimposed upon the former and which, on account of its appearance, was interpreted as being a normal aortic arch. The opacity was seen definitely to pulsate on fluoroscopy, and this was believed to indicate an enlargement of the aorta as well as of the left subclavian artery.

Examination of the lung fields failed to show any pathologic changes.

In May 1951, *thoracic aortography* was performed. The procedure was as follows: A No. 10 F catheter was introduced into the right radial artery, until its tip reached the middle third of the ascending aorta, when the injection was made, about 65 ml of Umbradil being administered within less than two seconds. No deformity of the ascending aorta or of the middle part of the arch was revealed. The vessels branching off from the latter also presented normal appearances. The distal part of the aortic arch and the upper portion of the descending aorta, however, were found to follow an anomalous course. At that site, the vessel was seen to form an arch with its concavity, and not its convexity, directed cephalad and backwards, the point of maximum curvature being at the level of the ligamentum arteriosum Botalli. No narrowing of the lumen was found

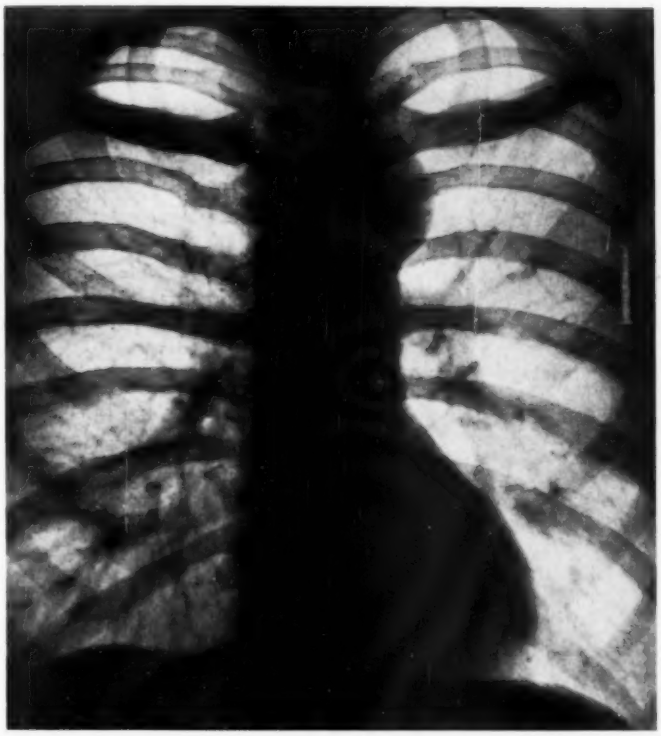

Fig. 1. Case 1. P.a. chest roentgenogram showing a semispherical, homogeneous opacity of soft-tissue density, sharply defined in contour, projecting beyond the upper left border of the mediastinal shadow. The opacity appears to be superimposed upon a roundish shadow of greater density, situated inferiorly.

to occur in that portion of the vessel. In front of the point of maximum curvature, there was a small, cone-shaped image filled with contrast medium. The lateral convexity, as seen in conventional roentgenograms, represented the distal part of the arch (Fig. 2).

The internal mammary and the intercostal arteries appeared to be normal in size.

The brachial and femoral blood pressure readings were as follows: Right arm: 110/70, left arm: 120/80; right leg: 150/90, left leg: 150/100.

The pedal arteries could be palpated in both limbs.

Pulse tracings taken at the level of the lower third of the legs, under resting conditions, showed a decrease in the amplitude of pulsations. The same finding was obtained at the wrists, especially on the right side.

In the other case the aortic anomaly, although morphologically similar to the one observed in the preceding case, had failed to bring about changes in the mediastinal shadow as apparent as in Case 1. The

a b

Fig. 2. Case 1. Thoracic aortography. a. P.a. The opacity observed in the conventional chest films is shown to correspond to the distal part of the aortic arch. The rounded area of increased contrast situated inferiorly is formed by the proximal portion of the thoracic aorta in orthoprojection. b. Lateral. The distal part of the arch and the proximal portion of the descending aorta exhibit a curvature the concavity of which is directed cephalad and backwards — an appearance very much like the one seen in coaretation. No signs indicative of stenosis present. The small, cone-shaped area on the medial border of the aortic shadow is most likely to be a remnant of the ductus arteriosus Botalli.

malformation was disclosed by an angiocardiographic examination to which the patient had been subjected following a clinical diagnosis of congenital heart disease.

Case 2. M. C. R., aged 12. Soon after birth, a 'cardiac bruit' had been detected on auscultation. The patient had never had dyspnoea or cyanosis.

Physical examination showed a normally developed subject. Heart: A systolic thrill could be felt over the whole cardiac area. On auscultation, a loud, harsh systolic murmur was heard, having its point of maximum intensity in the fourth left intercostal space

Fig. 3. Case 2. Selective angiocardiography. The distal part of the arch and the proximal portion of the descending aorta appear to be displaced markedly anteriorly.

along the border of the sternum, but audible all over the cardiac area. The systolic murmur masked the cardiac sounds. Blood pressure: 120/80.

Roentgen findings: Total volume was 430 cc, corresponding to 380 cc per square meter of body surface. There was enlargement of the heart, chiefly of the ventricles. The trunk of the pulmonary artery was not visible. The hilar and the pulmonary peripheral vessels were enlarged.

Cardiac catheterization: The pressure in the right ventricle was a little higher than in the pulmonary artery. In other respects no reliable evidence as to the existence of any pathologic changes was provided.

Angiocardiography: A No. F 9 catheter was introduced into an antecubital vein, and its tip directed to reach a point just below the pulmonary orifice. Forty (40) ml of Umbradil 70 % were injected. The pulmonary conus was visualized and found to be smaller than normal, whereas the portion of the common trunk situated close to the conus appeared to be slightly larger than normal. This amounts to saying that the infundibulum of the pulmonary artery was relatively stenosed. During the injection, some of the contrast medium was seen to pass through a defect in the upper interventricular septum into the upper part of the left ventricle in the neighbourhood of the aortic orifice, and then up into the aorta. The radiopaque medium subsequently passed into the pulmonary circulation, and thence to the left auricle and ventricle, and to the aorta, which became successively visualized. In addition, simultaneous visualization of the pulmonary artery was seen to occur, the contrast medium being shunted to that

vessel through the previously mentioned septal defect. The patency of this defect appeared to be rather wide.

In the lateral roentgenograms (Fig. 3), the posterior part of the aortic arch appeared to be displaced in a manner similar to that occurring in coarctation, *i. e.* that part of the vessel was displaced markedly forwards, with the point of maximum inflection at the attachment of the ligamentum arteriosum.

No evidence of stenosis could be found. The portion of the descending aorta situated immediately below the kinking, appeared to be larger than normal.

Discussion

Two instances of a rare congenital malformation of the distal part of the aortic arch and of the contiguous portion of the descending aorta are described, which, with those so far reported in the literature, bring the number of known cases up to eight.

This condition, is, however, probably far less uncommon than might be expected, if one realizes that (1) it is still little known; (2) it does not always entail appreciable mediastinal changes and, (3) on account of the fact that there is no stenosis, its clinical picture may be associated with very few signs and symptoms, or none at all. In fact, in our Case 2 the aortic abnormality was discovered accidentally owing to the co-existence of other congenital malformations.

The anomaly is characterized by deformation as well as elongation of the thoracic aorta.

The deformation chiefly affects the distal part of the arch and the adjoining first portion of the descending aorta, as has already been outlined in the preceding section of this paper. However, the other portions of the arch, and, to a lesser extent, the descending aorta, are involved as well.

According to JÖNSSON, BRODÉN and KARNELL, "the chief difference in course between the normal aorta and the aorta with coarctation lies in the fact that the stenosed area and the adjoining parts of the aorta are situated further towards the front. The arch of the aorta in particular is affected by this malposition."

This basic difference from the normal aortic arch existed both in our cases and in those reported by SOUDERS and co-workers, as well as in those described by DOTTER and STEINBERG.

The one feature distinguishing this anomaly from true coarctation lies in the absence of aortic stenosis, which in its turn results in the absence of the whole clinical picture associated with stenosis, *i. e.* left ventricle hypertrophy, collateral circulation, rib erosions, and so forth.

As regards elongation proper, this may be encountered in varying degrees. Thus, a very marked elongation was found in our Case 1, in SOUDERS and co-workers' Case 1, and in Case 2 of the series reported

by DOTTER and STEINBERG. By contrast, elongation was seen to be present to a minor degree in our Case 2, and in Case 1 of DOTTER and STEINBERG.

The site where elongation is especially prominent is the distal part of the aortic arch, whereas in the proximal portion of the descending aorta it is far less pronounced. It is this feature (*i. e.* elongation), which results in the appearance of a rounded upper mediastinal mass; evaluation of the eight cases so far reported in the literature appears to indicate that, in general, the more marked the elongation of the distal part of the arch, the larger is the opacity which is seen to project beyond the left border of the upper mediastinal shadow.

The clinical picture is neither uniform nor contributory. Thus, a systolic murmur was heard over the apex in the first of our cases, whereas a faint systolic murmur just median to each scapula was elicited by auscultation in SOUDERS and co-workers' Case 2; furthermore, a loud murmur, with its point of maximum intensity in the second left intercostal space and conducted to the clavicle, the back, as well as to the aortic area, was heard in their Case 3.

These systolic murmurs, being audible over the base of the heart as well as at the back over the interscapulo-vertebral region, may reasonably be presumed to be produced at the level of the aortic deformation.

The diagnosis may be a matter of considerable difficulty, if use be made solely of conventional roentgen procedures. The finding of the rounded area of increased density superimposed upon the opacity projecting from the upper left border of the mediastinal shadow may be of some aid. This appearance, which we found to be present in the postero-anterior conventional chest roentgenogram of our first case, corresponds to the first portion of the thoracic aorta in orthoprojection. Diagnosis, however, is largely dependent upon contrast visualization procedures, and especially upon thoracic aortography.

As far as the pathogenesis of this anomaly is concerned, SOUDERS and his associates believe it to be the same as that of coarctation. With respect to the latter, they share the view that the condition is due to the traction exerted on the isthmus of the aorta by an abnormally short ductus arteriosus. In discussing their cases, they suggested that the aortic deformity had been caused by traction, "the pull of which had been approximately uniform about the aorta, so that little tenting of the medial wall had been produced, and instead a uniform, tortuous double curvature of the distal aortic arch had occurred".

It should be stressed, however, that, at the present state of knowledge, the pathogenesis of coarctation is still obscure, so that the hypothesis offered by SOUDERS and his associates can hardly be said to be entirely satisfactory. Nevertheless, since the aortic deformity under

discussion closely resembles that of coarctation in character, it is likely that the pathogenetic mechanisms underlying both conditions are very much alike. It remains to be ascertained why in these cases no stenosis of the vessel has ensued.

SUMMARY

Two cases of a rare congenital abnormality of the aortic arch and of the adjoining first portion of the descending aorta are presented, the observations bringing the number of cases so far reported to eight. The clinical as well as the roentgenologic features of the anomaly are discussed, and the significance which may be attached to the condition, especially from a roentgenologic standpoint, is emphasized.

ZUSAMMENFASSUNG

Zwei Fälle mit einer seltenen kongenitalen Anomalie des Aortabogens und des angrenzenden ersten Abschnittes der Aorta descendens werden geschildert. Zusammen sind damit bisher 8 derartige Fälle beobachtet worden. Sowohl die klinischen als auch die röntgenologischen Befunde bei dieser Anomalie werden besprochen. Die Bedeutung, welche diesem Zustand besonders vom röntgenologischen Gesichtspunkt aus zuzuschreiben ist, wird hervorgehoben.

RÉSUMÉ

Les auteurs présentent deux cas d'une anomalie congénitale rare de l'arc aortique et de la première portion adjacente de l'aorte descendante, ces observations portant à huit le nombre des cas publiés jusqu'à maintenant. Les auteurs examinent les caractères cliniques et radiologiques de cette anomalie et soulignent l'intérêt que l'on peut attacher à cette malformation, particulièrement d'un point de vue radiologique.

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Reich: Quoted by Souders, Pearson and Adams.

FROM THE DEPARTMENT OF RADIOTHERAPEUTICS (DIRECTOR: PROFESSOR J. S. MITCHELL), UNIVERSITY OF CAMBRIDGE, ENGLAND

COMPARISON OF CYTOLOGIC EFFECTS PRODUCED BY 220 kV_p ROENTGEN RAYS, RADIO-IRIDIUM Ir^{192} GAMMA RAYS AND 30 MeV_p ROENTGEN RAYS IN THE MICROSPORES OF TRADESCANTIA BRACTEATA

by

*K. C. Bora**

A number of experiments have been carried out at the Radiotherapeutic Centre at Addenbrooke's Hospital, Cambridge in order to obtain further and more precise information on the relative biologic efficiencies of higher energy roentgen rays. The filtered and pulsed 30 MeV_p roentgen rays produced by the synchrotron have been compared with filtered 220 kV_p roentgen rays from a Maximar therapy machine and gamma rays of mean energy 0.4 MeV produced by radio-iridium Ir^{192}. The biologic criteria were cytologic changes in the microspores of *Tradescantia bracteata*. A preliminary account was given in the paper by MITCHELL, SMITH, ALLEN-WILLIAMS and BRAAMS, Part III (1953).

Material and Method. Young shoots of *Tradescantia bracteata* (*Clone 20[2] and 21[3]*) were used in all the experiments. The shoots were collected nearly 24 hours before the commencement of irradiation and kept in a control chamber which was continuously illuminated with white light. The temperature was controlled at 24° C ± 1 and the relative humidity was about 90 %. Under these conditions the shoots appeared to remain in good condition for about five days.

At the time of irradiation the shoots were placed either in a perspex tank or chamber containing water at 24 to 25° C. About ten minutes after irradiation they were transferred to the control chamber and remained there until the time of fixation of the smears of the irradiated anthers in 1 : 3 acetic-alcohol. The smears were then stained by Feulgen's method. The fixations were made at 24 and 72 hours after irradiation with a maximum variation of 10 minutes either way. For further details

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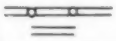
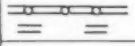
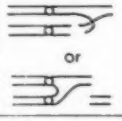
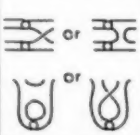
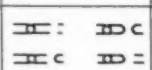
Type of aberration	Diagram	Number of breaks	Number of Reunions		Symbols
			actual	counted	
Dicentric		2	2	1	$C_2^{''}$ $C_0^{''}$
Tricentric		4	4	2	$C_3^{''}$ $C_0^{''}$
Monocentric ring		2	2	1	$C_r^{''}$ $C_0^{''}$
Triradial		2	2	1	Tr.
Chromatid Inter- or Intra-change		2	2	1	C.I.
Simple Chromosome break		1	–	–	$B^{''}$
Simple Chromatid break		1	–	–	$B^{'}$
Minutes		Not classified into breaks or reunions			m.

Diagram 1. Chromosome and chromatid aberrations.

of the cytologic method and the method of scoring the cytologic aberrations reference may be made to a previous paper (BORA 1954).

Types of Chromosome Aberrations. Chromosome aberrations produced by irradiation 72 hours before metaphase of first pollen grain mitotic division are predominantly of the chromosome type and have been described fully elsewhere (BORA 1954); those produced 24 hours before metaphase are mainly of the chromatid type. The chromatid reunions usually observed are symmetric or asymmetric inter- or intrachanges, both abbreviated C. I. triradials (Tr), and isochromatid breaks (B''), with or without sister reunion. The various types of aberrations examined in the microspores are presented in Diagram 1 and include 100 % chromosome and more than 95 % chromatid aberrations.

The *physical* details for the irradiation of the shoots are set out in Table 1.

Table 1

Physical conditions for irradiation of shoots of Tradescantia bracteata

Experimental series number	Measured mean dose rate at position of shoots, r per minute and 'r' per minute for 30 MeV_p roentgen rays	Dose rate at shoots in r and 'r'	Filtered 220 kV_p roentgen rays from Maximar unit			Radio-iridium Ir^{192} 0.4 MeV gamma rays, continuous radiation	Filtered 30 MeV_p roentgen rays from synchrotron-pulsed radiation
			Added filter (and mA)	H. V. L. of primary radiation mm Copper	S. S. D. d_1 and d_2 cm*		
1 a	3.16—3.26	300—307	2.5 mm Cu	2.55	123	24.3	83.7
1 b	3.26—3.3	150, 300	+ 1 mm Al (10 mA)		d_1=117 d_2=6.1	d_1=22.2 d_2=2.1	d_1=79.0 d_2=4.7
2	15	100, 200, 300, 400 and 500	0.64 mm Sn +2 mm Cu+ 1 mm Al	3.6	26.1 d_1=20.0 d_2=6.1	8.2 d_1=6.1 d_2=2.1	51.0 d_1=46.3 d_2=4.7
3	8	100, 200, 300, 400, 500 & 600 and 500, 600, 700, 800, 900 & 1,000	(15 mA) 2 mm Cu+ 1 mm Al (10 mA)	2.2	51.7 d_1=47.0 d_2= 4.7	(a) 12.7—12.0 d_1=7.2 d_2=5.5—4.8 (b) 14.6—14.3 d_1=7.2 d_2=7.4—7.5	(1) 88.7 d_1=84.0 d_2=4.7 (2) 82.7 d_1=78.0 d_2=4.7

* S. S. D. — Source Shoot Distance; d_1 — Distance from the source to the front wall of the perspex tank or chamber; d_2 — Distance from the anterior face of the front wall to the position of shoots in the water in the tank or chamber.

A. 30 MeV_p Synchrotron

The copper conical absorbing filter was used for flattening the isodose curves. In all the experiments the buds to be examined were placed 4.7 cm away from the anterior face of the front wall of the perspex tank. At this distance the 95 % isodose curves could cover an area of about 5 cm diameter. For measuring the dose a Victoreen chamber was immersed in the position to be occupied by the buds. The roentgen radiation from the synchrotron is produced in main pulses at the rate of 50 per second. Each main pulse is of a duration of about 100 microseconds and consists of about 50,000 short pulses each of a duration of about 1×10^{-9} seconds due to bunching of the circulating electrons.

The arrangements of shoots for each separate series of experiments were as follows.

Series a (*1952*). These experiments covered a period from 31 July 1952 to 2 August 1952. The shoots of *T. bracteata Clone 20*[2] were tied together in a small bundle, about 3.5 cm in diameter, placed in a glass jar, and kept in position in the perspex tank containing water at 25° C. The doses used were 150 and 300 r. The maximum variation in the dose was believed to be about $\pm$ 10 %.

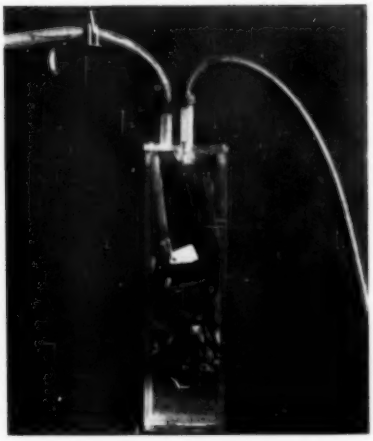


Fig. 1. Perspex chamber containing the shoots.

Series b (1953). These experiments were planned to use a higher dosage rate (15 r per minute) and as many different doses as possible. Since it was not possible to obtain 15 r per minute outside the collimator, the shoots (*T. bracteata Clone 21*[3]) were placed in a perspex chamber (12.5 × 5.3 × 5.3 cm, Fig. 1) containing water at 25° C. The chamber was then placed inside the collimator. The variation in the temperature was about ± 0.5° C during irradiation. These experiments covered a period from 17 to 23 September 1953.

Series c (1954). The dosage rate in this series was 8 r per minute and the shoots, *T. bracteata Clones 20*[2] and *21*[3], were placed outside the collimator. The perspex tank was fitted with a sliding partition with a hole, 5 cm in diameter, at the centre. This device was found to be very helpful in placing the buds more accurately at the desired position and was used in all the other experiments. The tank was lined up by sighting through the hole of the partition. The maximum possible error in the S. S. D. would be about ± 0.25 cm. As far as 'across the beam' is concerned, the possible error could be about ± 0.25 cm. The isodoses show a variation over ± 2.5 cm not greater than ± 3 % in the horizontal, and about the same, ± 3 % over ± 2.0 cm in the vertical. These experiments were carried out during the periods from 17 May to 23 June 1954, and from 11 to 16 June 1954. During the first period, the S. S. D. was 88.7 ± 0.25 cm. When the output of the machine fell below the required level it was found necessary to move the whole tank 6 cm nearer the target. The S. S. D. remained at 82.7 cm until the last experiment.

B. Radio-iridium Teletherapy Unit

The same perspex tank containing water at the required temperature was used for all the experiments carried out with this unit; Fig. 2 shows the experimental set up. It is of interest to note that the S. S. D. for Ir^{192} was much shorter than those of the other two machines and since it was practically impossible to arrange all the probable buds exactly at the same vertical plane facing the gamma ray beam, the variation in the dose received by different buds is believed to be greater than in roentgen ray

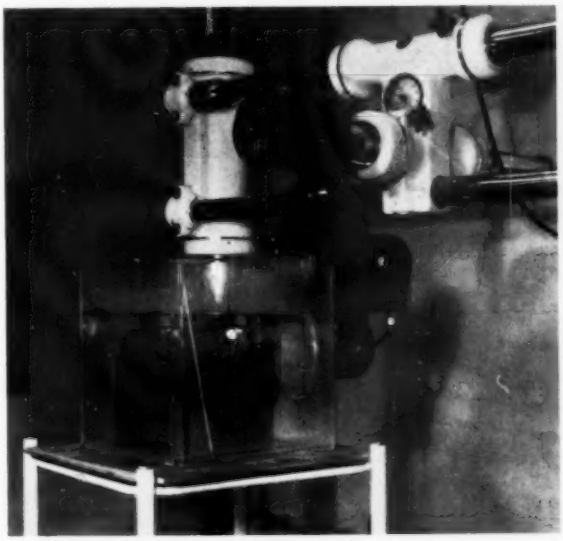


Fig. 2. Experimental arrangement for irradiation of the plant material, showing the perspex tank containing water and, in this case, irradiation with gamma rays produced by a source of radio-iridium, Ir^{192}, in a teletherapy unit.

doses. During the last series of experiments the radio-iridium was replaced on 11 June 1954 and consequently the S. S. D. for the period from 11 to 16 June 1954 was from 7.4 to 7.1 cm. Owing to the continuous decay of Ir^{192} with its half-life of 70 days, the S. S. D. had to be altered for each dose and the buds placed nearer the target each day. (Table 1, value of d_2 for gamma rays.)

C. 220 kV_p Maximar Unit

The same perspex tank was used for the irradiations of shoots with this machine. Other details are presented in Table 1.

For each dose the irradiations with the three machines were carried out in most cases on the same day. On some occasions, 2 to 3 experiments, each consisting of three irradiations, were carried out on the same day.

Experimental Data

The results of the first series of experiments (1951) are summarized in Tables 2 and 3. They show that, with equivalent doses, 220 kV_p roentgen rays produces more chromatid and chromosome aberrations and consequently more damaged microspores than either 0.4 MeV gamma rays

Table 2

Doses: 300—307 r; radiation intensity: 3.16—3.26 r per minute; time of fixation: 72 hours; temperature: 24 ± 1° C; material: Tradescantia bracteata 20²

Dose in roentgen	Type of radiation	Energy	No. of microspores observed	% of damaged microspores	*Aberrations* per 100 microspores					
					Total chromosome reunions ($C_2''C_0''$ + $C_3''2C_0''$ + C_1'' r C_0''	Simple breaks B''	B'	Total chromosome breaks	Minutes m	$B''+m$
300	roentgen	220 kV_p	450	50.44 ± 3.34	28.00 ± 2.49	4.44 ± 0.99	11.77 ± 1.61	60.44 ± 3.66	20.66 ± 2.14	25.11 ± 2.36
270—300	roentgen	30 MeV_p	600	31.00 ± 2.27	14.83 ± 1.57	2.33 ± 0.62	7.33 ± 1.10	32.00 ± 2.30	10.16 ± 1.30	12.50 ± 1.41
300	γ	0.4 MeV	800	33.12 ± 2.03	16.12 ± 1.41	3.37 ± 0.64	9.00 ± 1.06	35.62 ± 2.11	9.75 ± 1.10	13.12 ± 1.28

from Ir^{192} or 30 MeV_p roentgen rays from the synchrotron. A microspore has been considered damaged if it carries any form of chromosomal damage in any part of the six haploid chromosomes. It is of interest to note that there is no significant difference between the latter two radiations in the production of any type of chromosomal injury.

The data derived from the second series of experiments are presented in Diagram 2. The free-hand curves fitted to the experimental points represent approximate mean values of the aberration frequencies. The standard errors shown by the vertical lines have been calculated from the formula used previously (BORA 1954). As in the previous experiments, no significant statistical difference has been observed between the yields of chromosomal aberrations produced by the equal doses of 0.4 MeV gamma rays from Ir^{192} and 30 MeV_p roentgen rays from the synchrotron with the exception of the dose of 400 r. A freehand curve has therefore been drawn to represent the mean aberration frequencies of these two radiations. 400 r of 30 MeV_p roentgen rays produced anomalously low frequencies of chromosomal aberrations of all types and the total yield is equal to that produced by 300 r of the same radiation. Since the control measures were indentical in all the experiments and no conscious selection of material for examination was made, the biologic meaning of this variation was not understood and the experiment was repeated the following year. It may be emphasized that this particular dose was the first of the three doses given on the first day of irradiation. Nothing unusual was observed in the mannèr in which the buds received the dose. It is not yet known whether the fact that this was the first dose can provide

Table 3

Doses: 150 & 300 r; radiation intensity: 3.26—3.3 r per minute; time of fixation: 24 hours; temperature: 24 ± 1° C; material: Tradescantia bracteata 20²

Dose in roentgen	Type of radiation	Energy	No. of microspores observed	% of damaged microspores	*Aberrations*, per 100 microspores				
					Total chromosome reunions $C_2''C_0''$ + C. I. + Tr.	Simple breaks B''	B'	Total breaks chromosome + chromatid	Minutes m
150	roentgen	220 kV_p	565	66.72 ± 3.43	12.74 ± 1.50	26.90 ± 2.18	45.48 ± 2.83	97.87 ± 4.08	9.38 ± 1.26
135—150	roentgen	30 MeV_p	640	38.43 ± 2.45	3.59 ± 0.74	14.85 ± 1.52	26.56 ± 2.03	48.59 ± 2.75	3.91 ± 0.77
150	γ	0.4 MeV	480	42.50 ± 2.98	4.58 ± 0.97	17.70 ± 1.91	31.66 ± 2.56	58.54 ± 3.49	3.33 ± 0.83
300	γ	0.4 MeV	480	64.78 ± 3.67	17.50 ± 1.90	36.66 ± 2.76	48.75 ± 3.18	120.41 ± 5.00	11.04 ± 1.51

any satisfactory reason for the discrepancy observed. The rest of the data are in general agreement with the results of the previous experiments.

Altogether 12 doses were used in the last series of experiments to illustrate more fully the biologic effect produced by the radiations concerned. The doses were divided into two groups of 100 to 600 r and 500 to 1,000 r. All the experiments consisting of 36 treatments were distributed over a period of 18 days in order to exclude any appreciable seasonal variation as reported previously (BORA, 1955). Two chief points of interest in this series rest in the repetition of the 400 r dose and the inclusion of the high dose series. The results are graphically represented in Diagrams 3 and 4. Free-hand curves have been fitted for the aberration frequencies of the low dose series. As in the previous experiments, no significant difference has been observed between 30 MeV_p roentgen rays and 0.4 MeV gamma rays, again with the exception of the 400 r dose. It is very remarkable that this particular dose should show lower aberration frequency and the magnitude of reduction should be almost the same in both the series *i. e.* aberration frequency is equal to that produced by about 300 r of the same radiation. It may be noted that, in this series, *Clone 20²* was used instead of *Clone 21³*. A special effort was made to utilise all the experimental material available and no selection of material was made. Incidentally, the 400 r dose of this series was the first dose in the series to be given. Detailed physical consideration shows that the maximum variation in any dose could be about ± 6 %. This would of course mean that the nominal 400 'r' of 30 MeV_p roentgen rays could

Table 4

Results of χ^2 tests on the frequencies of chromosome reunions

Doses: 600 to 1,000 r, *Clone 21*[3]

Dose in roentgens	Between radiations	D. F.	χ^2	Probability
1,000	Ir^{192} & 30 MeV_p	1	3.622	0.058
900	"	"	1.526	0.235
800	"	"	1.252	0.265
700	"	"	6.095	0.011
600	"	"	0.013	0.90

Table 5

Dose ratios for chromosomal effects of 220 kV_p and 30 MeV_p roentgen rays and gamma rays of mean energy of 0.4 MeV

Material	Doses	Type of aberrations	Range of doses bc_m/a	ratios	Average doses bc_m/a	ratios
(a)		CR	1.45—1.66		1.54 ± 0.08	
		TCB	1.43—1.53		1.48 ± 0.04	
		DM	1.40—1.47		1.42 ± 0.03	
T. bracteata	100–500r	B"	1.42—1.53		1.47 ± 0.05	
Clone 21[3]		m	1.50—1.57		1.53 ± 0.09	
		B" + m	1.42—1.47		1.44 ± 0.02	
			b/a	c/a	b/a	c/a
(b)		CR	1.134—1.149	1.219—1.261	1.140 ± 0.009	1.241 ± 0.015
		TCB	1.134—1.176	1.229—1.314	1.156 ± 0.014	1.270 ± 0.035
T. bracteata		DM	1.142—1.180	1.257—1.290	1.169 ± 0.014	1.273 ± 0.012
Clone 21[3]	600—	B"	1.181—1.250	1.4281–1.498	1.225 ± 0.025	1.464 ± 0.034
	1,000 r	m	1.282—1.138	1.200—1.276	1.218 ± 0.052	1.240 ± 0.033
		B" + m	1.285—1.138	1.229—1.308	1.215 ± 0.051	1.269 ± 0.034
			bc_m/a		bc_m/a	
(c)		CR	1.24—1.53		1.40 ± 0.12	
		TCB	1.28—1.63		1.43 ± 0.12	
		DM	1.31—1.36		1.33 ± 0.02	
T. bracteata		m	1.26—1.80		1.43 ± 0.20	
Clone 20[2]	100—600	B" + m	1.32—1.61		1.48 ± 0.10	
	r	B"	b/a = 1.872—1.1875	c/a = 1.685—1.764	b/a = 1.870 ± 0.007	c/a = 1.73 ± 0.035

Cr — Chromosome reunions; TCB — Total chromosome breaks; DM — Damaged microspores (a) — 220 kV_p roentgen ray dose; (b) — Gamma ray dose of mean energy 0.4 MeV; (c) — 30 MeV_p roentgen ray dose; bc_m — mean dose of b and c.

be as low as 376 'r' and the nominal 300 r of 0.4 MeV gamma rays or 30 MeV_p roentgen rays could be as high as 318 r.

It is highly improbable that this variation should be exhibited only by the first dose in each case. One is tempted to assume that some kind of hitherto unknown qualitative variation in the radiation might exist

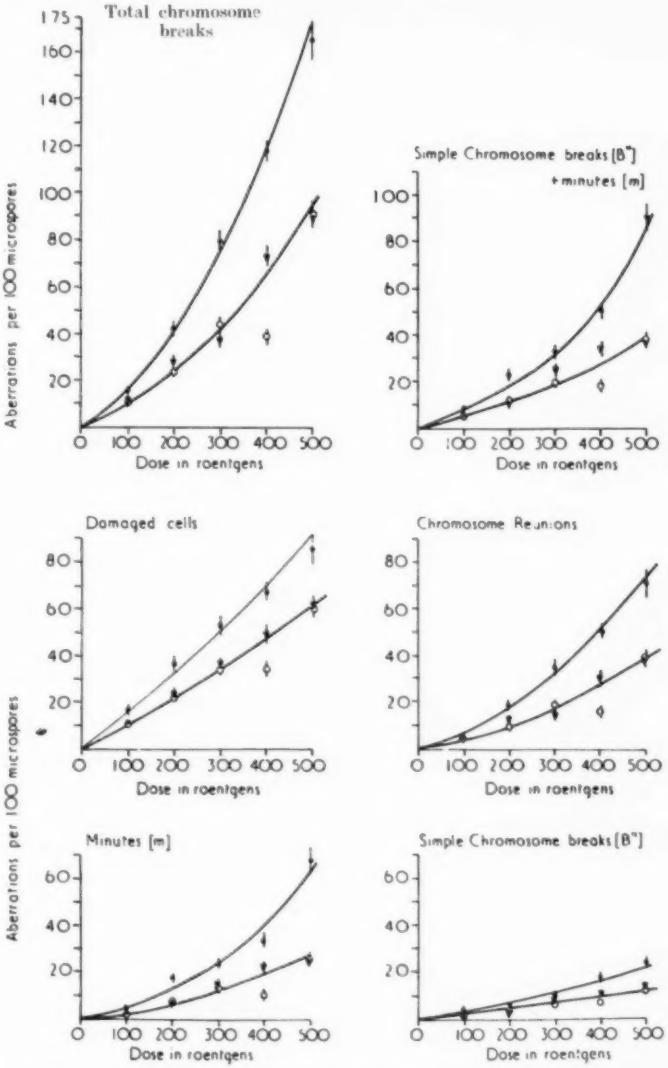


Diagram 2. Dosage-aberration frequency curves of 220 kV_p and 30 MeV_p roentgen rays and 0.4 MeV gamma rays. Doses 100 to 500 r; intensity 15 r per minute; fixation time 72 hours; temperature 25° C. *Tradescantia bracteata, Clone 21*[3].

● ● 220 kV_p roentgen rays.
–○ ○– 30 MeV_p roentgen rays.
◀ ▶ 0.4 MeV gamma rays.

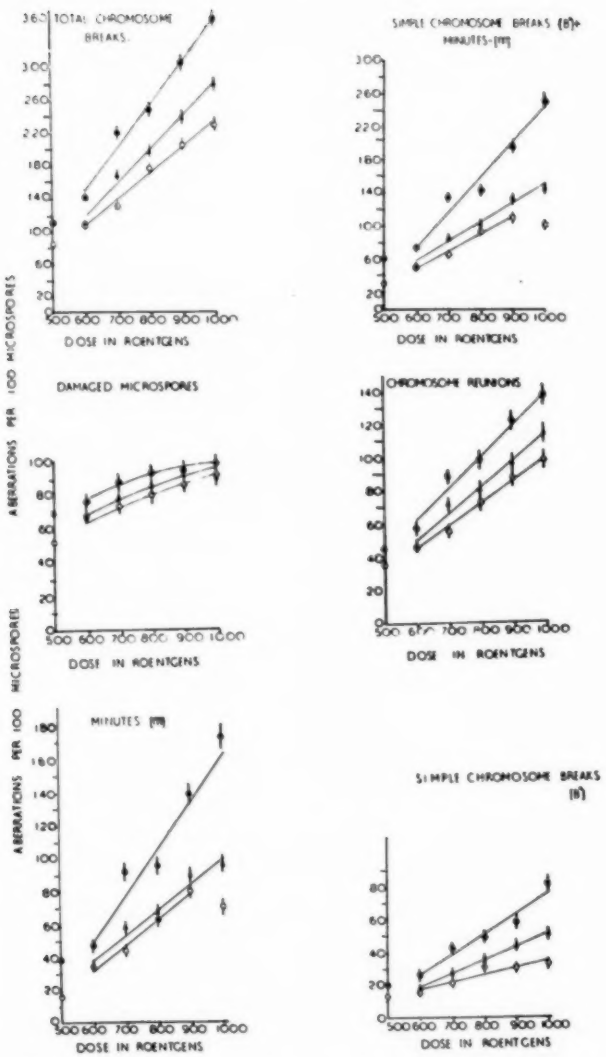


Diagram 3. Dosage-aberration frequency curves 220 kV_p and 30 MeV_p roentgen rays and 0.4 MeV gamma rays. Doses 600 to 1,000 r; intensity 8 r per minute; fixation time 72 hours; temperature 24 $\pm$ 5° C. *Tradescantia bracteata, Clone 21*[3].

around the region of 400 'r' and the biologic reaction is therefore different from the rest of the series. Even if it is assumed that maximum variation occurred in this particular case, a reduction of 24 r from 400 r alone would fail to account for the observed fall in the aberration frequency. Further experiments are in progress to find out whether the anomalous effect of this dose is genuine or not.

The results from the high dose series show a considerable departure from those of the low dose series. The effects of the equal doses of 30 MeV_p roentgen rays and 0.4 MeV gamma rays are no longer equal. Although statistically not significant (Table 4), the former radiations produced consistently low frequency of aberrations at a dose of 700 r or more. The aberration yields of these doses, except that of 500 r, suggested that straight lines could be fitted to the experimental points with reasonable accuracy. Regression lines have therefore been drawn to represent the mean aberration frequencies of the radiations. The errors shown by the vertical lines are not the errors of the regression lines but they are the sampling errors calculated by the method already mentioned above.

Of all the aberrations produced 72 hours before metaphase, the chromosome reunions could be scored with sufficient ease and with a high degree of accuracy. They also represent the major proportion of the aberrations observed. It seems to me that they are the most reliable of all the data presented here. The regression lines lie almost within the limits of the standard errors. The dose of 500 r has been excluded from the regression lines, firstly, because of the non-linear dosage-reunion relationship and, secondly, no material treated with Ir^{192} gamma rays was available for examination. A significant fall in the frequency of the minutes has been shown by 1,000 r of 30 MeV_p roentgen rays and for this reason they are also excluded from the calculated lines. Much attention should not be given to this fall because of the inconsistency in the occurrence of the minutes on some of the slides belonging to all the treatments. Minutes do not appear to be a reliable criterion for any type of investigation due to the difficulty in accurate scoring. Since almost all the microspores (93.6 ± 5.5) were damaged by a dose of 800 r of 220 kV_p roentgen rays, a dose higher than this could not make any difference in the frequency of the damaged microspores, due to the fact that a microspore has been considered damaged if it carries any form of chromosomal injury. With doses higher than 800 r, the frequency of the chromosomal aberrations will be increased but not that of the damaged microspores. Mean free-hand curves have been drawn to represent the frequencies of damaged microspores. Another point of interest in this series is the smaller difference between the aberration frequencies of 220 kV_p roentgen rays and those of the other two radiations.

The doses of the three radiations to produce equal cytologic effects may be obtained either from the curves or the regression lines, and for each type of aberration measurements were made at 3 to 7 points. The ratio of the doses of any two radiations for producing equal biologic effects were calculated from the doses obtained from the curves or the regression lines. The average value of the readings has been taken to denote the dose ratio for the radiations concerned. The values of dose ratios for different radiations for producing various types of biologic effects are presented in Table 5 together with their range of variations and standard errors.

Discussion

The results suggest that the relative biologic efficiency (*rbe*) of roentgen and gamma radiations may apparently vary with the dose. A comparison of the results from the experiments with 100 to 500 r and 100 to 600 r tend to show that although these two experiments were separated by a period of one year and the clones used were different, all the values of *rbe* except those of B″ lie within the limits of experimental error. It is interesting to note that, despite the fact that a lower radiation intensity (8 r per minute) was used in the second series (100 to 600 r), the yields of aberrations are not smaller for equivalent doses than those of the other series (100 to 500 r) with a higher intensity (15 r per minute). According to the established dosage-intensity-reunion-frequency relationship a lower frequency of aberrations is expected from the doses with low radiation intensity. Strict comparison of these data is however not permissible owing to the facts already mentioned above, namely different times of the year and different clones. Both these experiments have already shown that at the dosage level used the dose ratios for 30 MeV_p roentgen rays and 0.4 MeV gamma rays are equal. For chromosome reunion their mean average values are 1.54 ± 0.08 and 1.40 ± 0.12 for two series with 100 to 500 r and 100 to 600 r, respectively.

These values are however considerably reduced at high dosage level. Taking chromosome reunions as a biologic criterion, it has been shown that for producing equal frequency of reunions at high dosage level, 30 MeV_p roentgen rays are 1.241 ± 0.015 times less efficient than 220 kV_p roentgen rays which in turn are 1.140 ± 0.009 times more efficient than 30 MeV_p roentgen rays. This of course means that 0.4 MeV gamma rays are about 1.10 times more efficient than 30 MeV_p roentgen rays. It is important to note that this difference is exhibited only when the dose is larger than 600 r. Results of t-tests made on the regression lines are given in Table 6. Although the total doses used for treating malignant tumours are usually large in comparison with the high doses used in these experiments, the average daily therapeutic dose is relatively smaller. It has already been

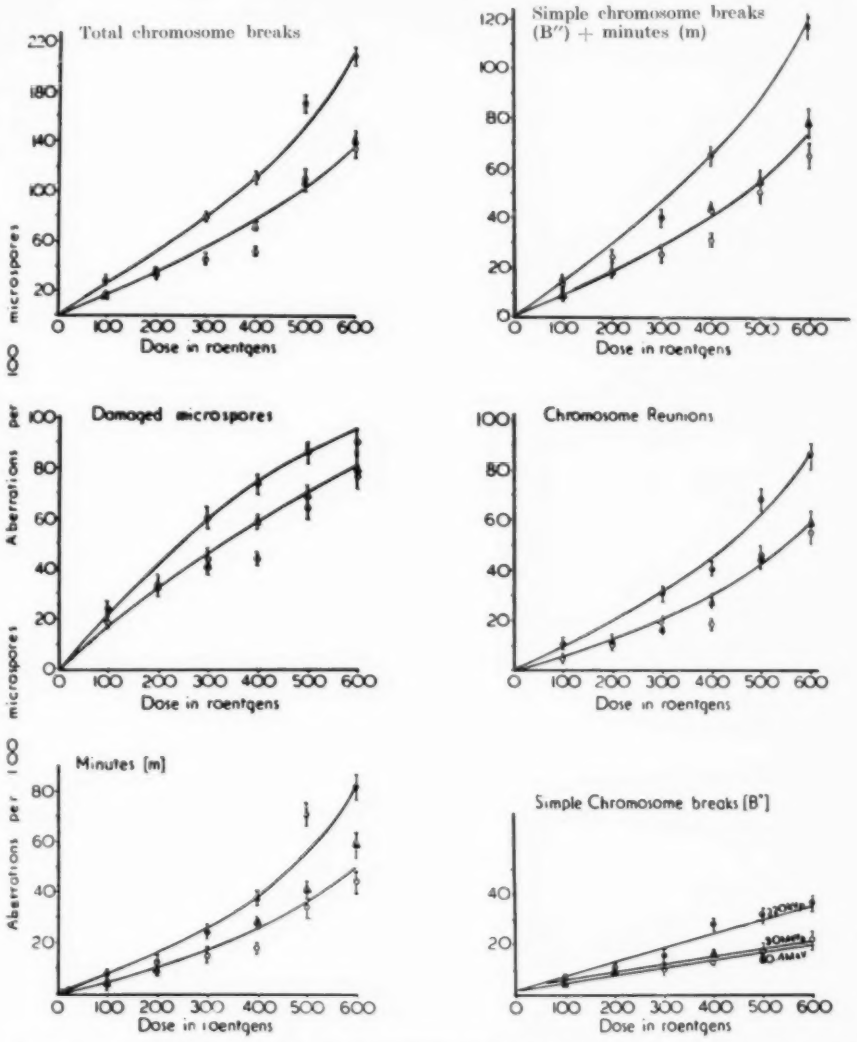


Diagram 4. Dosage-aberration frequency curves of 220 kV_p and 30 MeV_p roentgen rays and 0.4 MeV gamma rays. Doses 100 to 600 r; intensity 8 r per minute; fixation time 72 hours; temperature 24 ± 0.5° C. *Tradescantia bracteata, Clone 20*[2]. Total chromosome breaks: (Reunion breaks + simple breaks (B″).) Simple chromosome breaks (B″) + minutes (m).

Table 6

Test for significance of the difference between the two regression coefficients

Doses: 600 to 1,000 r, radiation intensity 8 r per minute; material: *Tradescantia bracteata, Clone 2P*.

Type of aberration	*Radiation*	*t*	*D. F.*	*Probability*
Reunion	220 kV_p roentgen rays & 0.4 MeV gamma rays	1.5976	6	P = 0.168
"	220 kV_p & 30 MeV_p roentgen rays	3.1068	6	P = 0.0205
"	0.4 MeV gamma rays & 30 MeV_p roentgen rays	1.8142	6	P = 0.130
Total chromosome breaks	220 kV_p roentgen rays & 0.4 MeV gamma rays	2.4292	6	P = 0.051
"	220 kV_p & 30 MeV_p roentgen rays	4.5829	6	P = 0.002
"	0.4 MeV gamma rays & 30 MeV_p roentgen rays	2.8145	6	P = 0.031
Simple chromosome breaks	220 kV_p & 0.4 MeV gamma rays	2.7469	6	P = 0.0335
"	220 kV_p & 30 MeV_p roentgen rays	4.4064	6	P = 0.003
"	0.4 MeV gamma rays & 30 MeV_p roentgen rays	3.7473	6	P = 0.0095

shown (BORA, 1955; DE SERRES and GILES 1953) that the cytologic effects of a fractional roentgen ray dose are significantly smaller than those produced by the same dose delivered continuously. It is often considered that there is a close correlation between cell death and chromosome damage. When high doses such as 1,000 r are used for radiocytologic investigations, the scoring of the aberrations with a high degree of accuracy becomes difficult. The values for high doses are therefore, to a certain extent relative and not absolute.

It is emphasized that with the present high dose series there was no real difficulty in scoring the chromosome reunions with reasonable accuracy and that no gross error is therefore introduced in regarding them as absolute values.

Radiation doses producing more than 75 chromosome reunions per 100 microspores cannot be used satisfactorily for the absolute value of total aberrations. The most reliable results could be obtained when the percentage of chromosome reunions lies between 20 and 75. In these experiments the most reliable values of the dose ratios are considered to be those obtained for doses from 300 to 500 r, and these correspond to an average value of about 1.4.

Acknowledgement

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SUMMARY

The relative biologic efficiency of filtered 220 kV_p roentgen rays from a Maximar therapy machine, gamma rays of mean energy 0.4 MeV from radio-iridium (Ir^{192}), and filtered and pulsed 30 MeV_p roentgen rays from the synchrotron, in producing chromosomal aberrations in the microspores of *Tradescantia bracteata* (*Clones 20² and 21³*) have been determined at several dosage levels. At low levels (100 to 500 r), equal doses of 30 MeV_p roentgen rays and 0.4 MeV gamma rays produce equal frequencies of chromosomal aberrations but, at higher dosages (600 to 1,000 r), the same dose of the former radiation produces consistently lower frequencies of all types of aberration.

ZUSAMMENFASSUNG

Die relative biologische Wirksamkeit filtrierter 220 kV_s Röntgenstrahlen einer Maximar-Therapiemaschine, Gammastrahlen einer mittleren Energie von 0.4 MeV einer Radioiridium (Ir^{192})-Teletherapieeinheit sowie filtrierter und pulsierender 30 MeV_s Röntgenstrahlen eines Synkrotrons für die Erzeugung chromosomaler Abweichungen der Mikrosporen der *Tradescantia bracteata 20² und 21³ Klon*) ist mit Dosen verschiedenen Grösse untersucht worden. In dem niedrigen Dosisgebiet (100 bis 500 r) erzeugen gleiche Dosen der 30 MeV_s Röntgenstrahlen und der 0.4 MeV Gammastrahlen die gleiche Anzahl chromosomaler Abweichungen, während dagegen in dem hohen Dosisgebiet (600 bis 1,000 r) dieselbe Dosis der ersteren Strahlung eine geringere Anzahl aller Abweichungsformen produziert.

RÉSUMÉ

Les efficacités biologiques de rayons roentgen filtrés de 220 kV_p émis par un appareil de thérapie Maximar, de rayons gamma d'énergie moyenne 0.4 MeV émis par un appareil de téléthérapie au radio-iridium (Ir^{192}), et de rayons roentgen filtrés et pulsés de 30 MeV_p émis par le synchrotron pour la production d'aberrations chromosomiales sur les microspores de *Tradescantia bracteata* (*Clones 20² et 21³*) ont été comparées pour plusieurs intervalles de doses. Dans l'intervalle inférieur de doses (100 à 500 r), des doses égales de rayons roentgen de 30 MeV_p et de rayons gamma de 0.4 MeV produisent des fréquences égales d'aberrations chromosomiales; mais dans l'intervalle supérieur de doses (600 à 1,000 r) la même dose de la première radiation donne lieu à des fréquences plus faibles de tous les types d'aberrations.

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FROM ROENTGEN DEPARTMENT II (DIRECTOR: DOCENT W. MAGNUSSON), SÖDERSJUKHUSET, STOCKHOLM, SWEDEN

ASCENDING PHLEBOGRAPHY IN VENOUS INSUFFICIENCY

by

Torgny Greitz

The upper part of the thigh and the pelvis have not been included in most phlebographic studies of venous disturbances of the leg. The few authors (LÖFSTEDT 1946, LINDE 1949, HÖJENSGÅRD 1951) who have published sizable material of ascending phlebographies of the lower extremities in the vertical position have all restricted their examinations to the lower part of the leg. SYLVAN (1951) and ÖDMAN (1952) have examined the veins of the thigh by means of retrograde phlebography in the erect position.

In the present material the extremities have all been explored from the foot to the pelvis. They have been examined in the vertical position with a uniform technique (GREITZ 1954). When it has not been possible to demonstrate patency of the external iliac vein in the routine examination, an additional pelvic phlebography has been made in accordance with the principles described in a previous paper (GREITZ 1955).

Material. 280 extremities have been examined. Of these, 124 were right legs and 156 left legs, the patients numbering 251, out of which 69 were men and 182 women, ranging from 15 to 82 years of age, the average age being 51. Seventeen cases had undergone previous operation with ligation of the long saphenous vein. Pelvic phlebography has been made in 59 cases.

The reason for the examination has generally been a suggestion of an earlier thrombosis. This suggestion has, however, been more or less well grounded; sometimes the surgeon has had the impression that the leg had a 'post-thrombotic appearance' despite the fact that the anamnesis gave no information about thrombosis; some cases have been referred because of an inexplicable edema; in other cases, the patients'

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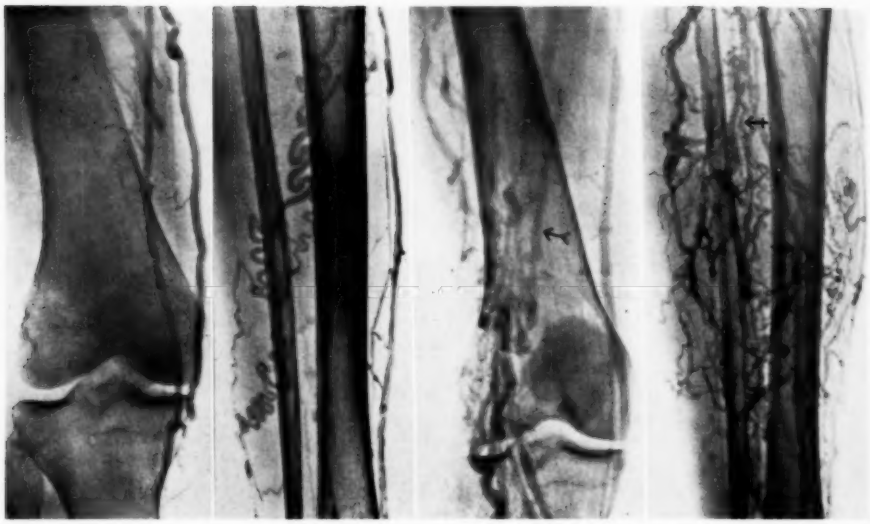


a b c d

Fig. 1. a, b. Clinical signs of acute thrombosis. Only the distal part of the anterior tibial vein is filled. The contrast medium passes through superficial veins in the lower part of the leg. In the mid thigh the profunda femoris vein is filled through anastomoses. c, d. 9 months later the deep veins are re-canalized. Note irregular lumina of posterior tibial (↑) and peroneal (↕) veins. At the site of the popliteal vein several irregular vessels are seen, one of which is the medial concomitant popliteal vein (↑↓).

statements have been interpreted to mean that there probably had been a superficial thrombophlebitis previously, but phlebography was demanded 'for safety's sake'. For these reasons I have considered it appropriate to depart from the usual classification of the material on clinical data. The difficulty of differentiating between primary and post-thrombotic varices from the history is illustrated by the following case.

A 39-year-old man, otherwise completely healthy and working, sought medical attention for an edema of the right leg, which had been increasing for two weeks. Phlebography resulted in contrast filling of the distal part of the anterior tibial vein, but otherwise no filling of the deep veins either in the calf or thigh. On further phlebography nine months later the peroneal, the anterior and posterior tibial, the popliteal, and the femoral veins, were all filled. They had, however, a somewhat irregular course and uneven contours, and — with the exception of the anterior tibial vein — lacked valves. (Fig. 1.)

There can be no doubt that this patient had suffered from acute thrombosis which had started without his having been confined to bed. It is also clear that such changes as were observed at the second examina-

a b c

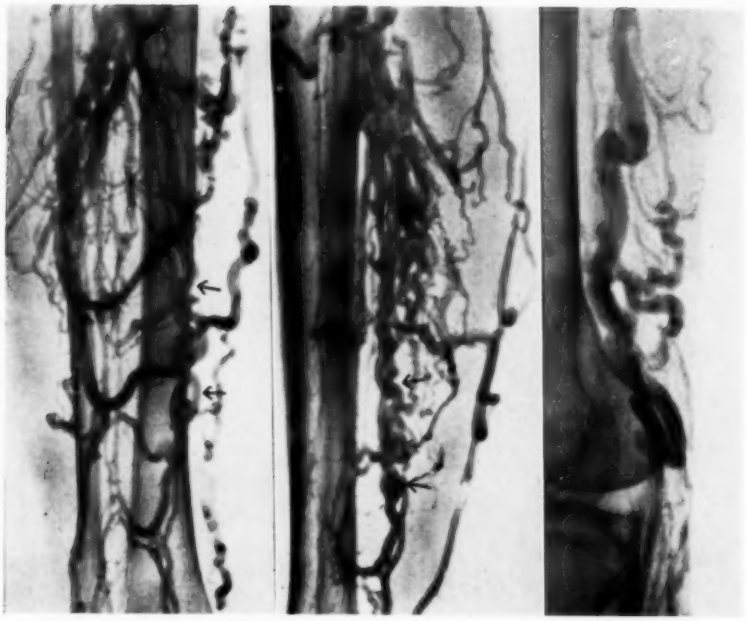
Fig. 2. a. Local dilatation of the popliteal vein. b. General dilatation of the same vein. Its course is less straight than usual. c. Local dilatation superimposed on general widening of the upper part of the popliteal vein.

tion may have been caused by thrombosis. This has been convincingly demonstrated by HÖJENSGÅRD (1951); patients with such changes are classified as one group in the division of the material. My classification is exclusively based on roentgenologic changes.

Fundamental pathologic changes and their existence in different veins

Organic changes of three different kinds have been observed in this material: dilatation, which may be either local or general; tortuosity, which in combination with dilatation is called varicosity; and finally post-thrombotic changes, which are characterized by uneven contours, irregular lumina and — sometimes — erratic coursing.

A. *Changes in the deep veins.* Dilatation of the deep veins, demonstrated by phlebography, has earlier been described by IMLER, BEAVER and SHEEHAN (1944), ANDERSSON and PATTERSON (1945), JENNY (1947), and BOYD (1948). These observations are otherwise interpreted by



a b c

Fig. 3. Varicose changes of the posterior tibial vein (a, b) and the popliteal vein (c).

OLIVIER (1950), who writes, "les veines profondes intramusculaires de la jambe sont le siège de dilatations normales qui ont été prises à tort pour des lésions pathologiques", and by DOW (1951), who states that "apparent dilatation of the deep veins may be within normal pathologic limits". It is true that the normal spindle shape of the deep veins, which may be very pronounced (GREITZ 1955), has often been confused with pathologic dilatation, although such a distension may undoubtedly occur. FELDER (1954), for instance, published an illustration which without a doubt shows 'a segmental dilatation' of a deep vein of the calf.

Local or general widening of the deep veins is in fact not unusual (Figs. 2, 3, 6). It may occur anywhere, even though the popliteal vein and the uppermost part of the femoral vein are predilected. A vein is rarely if ever quite uniformly dilated. The walls are therefore not completely parallel. The course of the vein is often not as straight as in normal cases (Fig. 2 c). A deep vein of the calf is not unfrequently dilated down to the level where it receives a widened communicating vein, while distally to that point it regains its normal calibre (Fig. 6).

The question whether the deep veins may be varicose or not, *i. e.* if they can be at the same time tortuous and dilated, has been discussed ever since 1855, when VERNEUIL described deep varices in an autopsy material. The existence of deep varices was, however, on the basis of anatomic studies, denied by REMY (1901), DELBET and MOCQUOT (1913), and MEISEN (1932). BERNTSEN (1925) found, on the other hand, one case with a varicose posterior tibial vein in the dissection of 21 extremities with varices. Varicose changes of the deep main veins are not found in phlebography according to COTTENOT and RAVINA (1933), OLIVIER (1950), DOW (1951), and HÖJENSGÅRD (1951). FELDER (1953) published one case, of which he says, "the dilatation and tortuosity of the deep veins is apparent". The appended illustration is, however, not quite convincing.

Varicose changes of the deep main veins are rare, and do not become as pronounced as in the superficial veins (Figs. 3, 6). They may, however, occur in all the deep veins, with the exception of the anterior tibial vein, which I have never observed to be varicose. The distal part of the posterior tibial vein behind the medial malleolus is in many cases of pronounced varices both tortuous and dilated, but this part of the vein is so close to the fascia that it can hardly be termed deep; it can best be compared to the subfascial part of the short saphenous vein. To sum up, there is no doubt that varices in the deep main veins do exist (Fig. 3), and this century-old discussion may thus be considered terminated.

The fact that post-thrombotic re-canalized veins have an irregular lumen and uneven walls is nowadays well known, and has been described by a number of authors: DOS SANTOS (1938), DOUGHERTY and HOMANS (1940), BAUER (1942), HÖJENSGÅRD (1949), DOW (1951), COCKETT (1953), and FELDER (1953). HÖJENSGÅRD, who alone among these has used the 'vertical technique', points out that the post-thrombotic veins lack valves and have a tortuous course (Fig. 4).

This tortuosity is sometimes caused by a segmental re-canalization of the double veins, segments on different levels being combined through pre-existing transverse anastomoses to form a tortuous trunk (Fig. 4 c). Tortuosity of the veins of the thigh may arise in a similar manner when the profunda femoris or the concomitant veins function as collateral channels (Figs. 10, 11).

B. *Changes in the muscular veins.* Varices in the muscular veins have been described by anatomists (among others, VERNEUIL 1855 and BERNTSEN 1925), but seem to have escaped the attention of radiologists. HÖJENSGÅRD (1951) writes: "The veins in the muscles of the leg have received very little attention hitherto. In patients with varicose veins ... they are often visualized, always in the form of valveless and tortuous, *i. e.* varicose, veins." This is, however, saying too much, since according

a b c

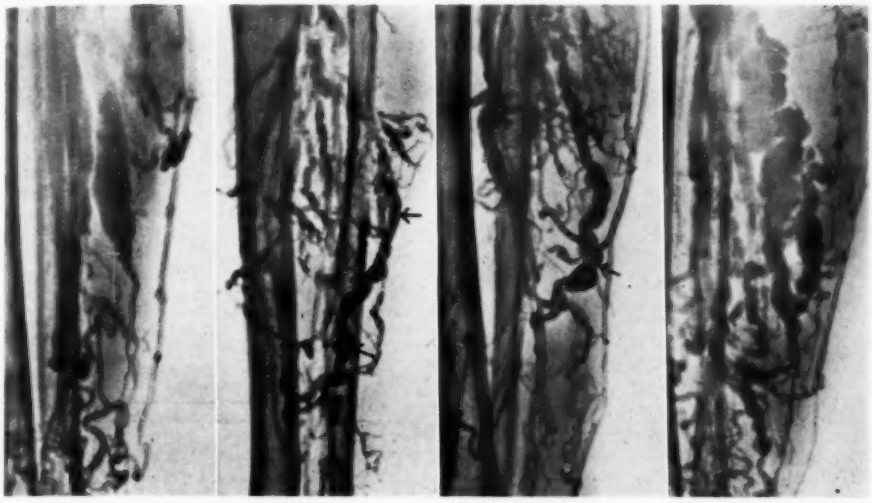
Fig. 4. Post-thrombotic limbs. a. Deep veins with irregular lumina lacking valves. b. Irregular lumina (↑). The posterior tibial vein has a somewhat tortuous course (↕). c. Tortuosity caused by segmental re-canalization of a double vein, segments on different levels being connected through transverse anastomoses.

to my experience it is not unusual to find completely normal spindle-shaped muscular veins in the calf even in cases with extensive varices. Sometimes the muscular vein may be partly normal, partly varicose. It is then generally the distal part of the arch formed by the vein that is varicose (Fig. 5 b, c). The distension of a muscular vein may become marked, but the degree of tortuosity seems to be more limited, due to the fact that such a vein cannot expand in the same manner as a superficial vein; the vein seems to be confined in its intramuscular space (Fig. 5 d). Varicose muscular veins are found also in the thigh (Fig. 9 c), as pointed out by SHERMAN (1944), HÖJENSGÅRD (1951), and SYLVAN (1951).

Post-thrombotic changes may also be observed in the muscular veins.

According to MARTORELL (1946), BAUER (1948), and HÖJENSGÅRD (1951), muscular veins filled in retrograde direction are incompetent. As I have previously emphasized, this is a normal finding (GREITZ 1955), which must not be considered pathologic.

C. *Changes in the communicating veins.* Retrograde filling of the communicating veins has also been interpreted as an indication of incompetence (MASSELL and ETTINGER 1948; MOORE 1949; MARTIN and



a b c d

Fig. 5. Dilatation of muscular veins. a. Highly dilated vein in the soleus. b, c. Varicose, *i. e.* tortuous and dilated muscular vein; changes most marked in its distal part. d. Pronounced varicosity of a soleus vein, which appears 'confined'.

McCLEERY 1950; VAN HOVE 1952). This is, however, also a normal occurrence (GREITZ 1955). Incompetence may, according to LINTON (1938) and SHERMAN (1944 and 1949), be determined from the morphologic changes demonstrable at autopsy. Distended communicating veins have been shown by phlebography by LESSER and RAIDER (1943), MARTORELL (1946), BAKER (1947), OLIVIER (1948), and DOW (1951). DOW points out 'that localized dilatation develops in the superficial vein at its junction with the communicating vein'.

A communicating vein may be tortuous without being distended (Fig. 6 a). It is true that when it is dilated, a distension is also present in the superficial vein which it drains. Not infrequently a more or less local widening also of the deep vein may occur. Both the deep and the superficial veins may often be seen to be of normal or only slightly increased calibre below the junction with the dilated communicating vein (Fig. 6 b). In this manner a 'communicating U-tube' is formed, the deeper limb of which may also be a muscular vein (Fig. 6 c). Marked changes of this type were found in almost one third of the extremities in my material.

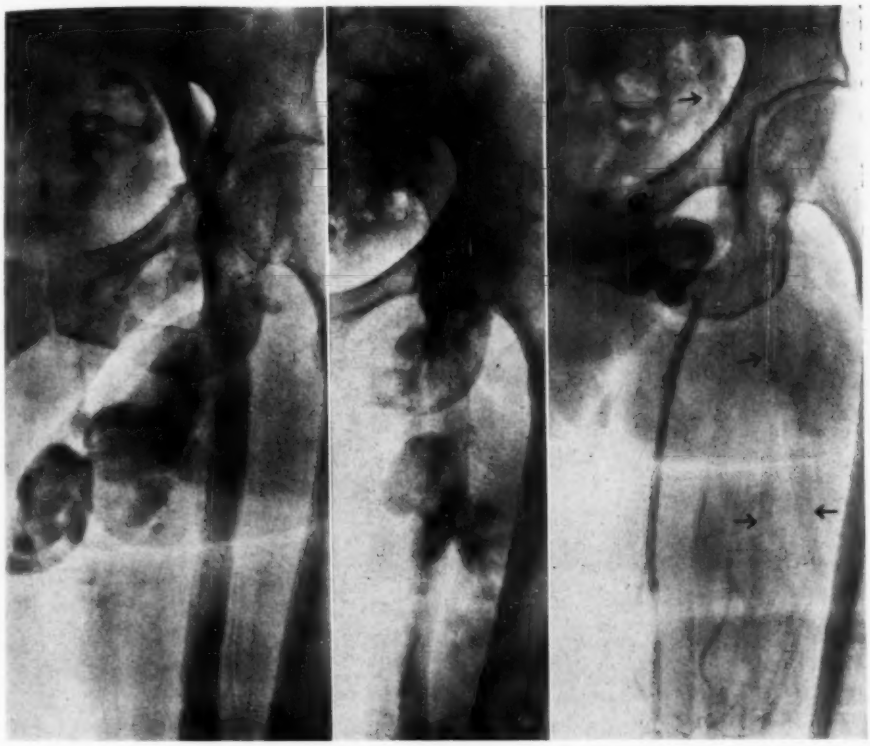
D. *Changes in the superficial veins.* The appearance of different kinds of varices has been described by authors since the days of varicography

a b c d

Fig. 6. a. Tortuous, but not dilated communicating veins. b. Dilated communicating vein (↑), connecting a deep (the posterior tibial) and a superficial (long saphenous) vein, which are dilated proximal to the junction, thus forming a 'communicating U-tube'. c. The deep limb of the 'tube' is a muscular vein (↑), the superficial one is the small saphenous vein (↑), which has a normal appearance distal to the junction with the dilated communicating vein. d. Widened anastomoses connecting deep veins.

in the 'thirties. In this connection I only want to emphasize that the phlebographic technique employed has been developed particularly with consideration to the deep veins, and that extensive superficial varices, although clinically demonstrable, have on many occasions failed to fill, particularly in the upper part of the leg. It is, however, possible to obtain filling of superficial varices even in the uppermost part of the thigh (Fig. 7 a—c). Varices have thus been observed in the pubic region in no less than 77 out of 280 extremities.

Venous spasm in phlebography has been stated to be a common phenomenon. ADAMEK and FRIEDLÄNDER (1936) report that a vein in spasm is concentrically narrowed, and that the constriction has a regular limitation. The illustrations of 'venous spasm' that have been published

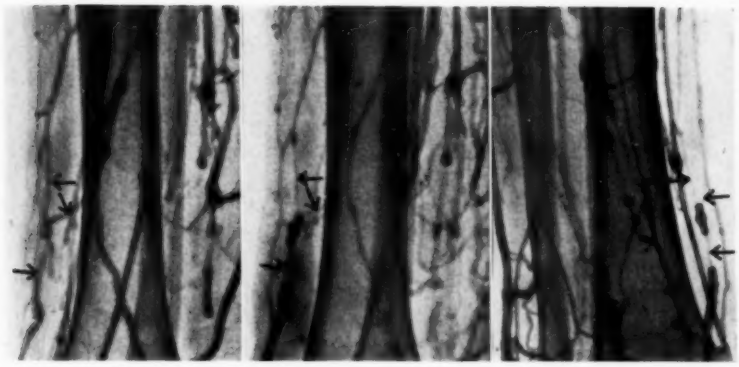


a b c

Fig. 7. Films of the pelvis obtained in routine examinations in erect position. a. Highly varicose long saphenous vein, filled in a retrograde direction. b. Recurrence after ligation of the long saphenous vein. c. In post-thrombotic limbs the superficial veins are often better filled than the deep ones (↑). Note varices in the pubic region.

(Lesser and Raider 1943; Papper and Imler 1944; Raymond, Adams and Mc Corriston 1948; Felder 1953) are in no way different from the appearances of false constrictions due to layer formation seen in normal veins examined in the horizontal position and with a vertical beam projection.

I have never observed spasm of the deep veins, but on a few occasions have seen changes in the calibre of a superficial vein which most likely have not been of organic origin and in appearance similar to the spastic contractions of the arteries of the brain in cases of subarachnoidal haemorrhages (Fig. 8).



a b c

Fig. 8. Spasm of a superficial vein (b, c), which in a previous examination had a uniform width (a). Both examinations made in a vertical position and with a horizontal projection; no layer formation or laminar flow observed. The second injection was partly perivenous.

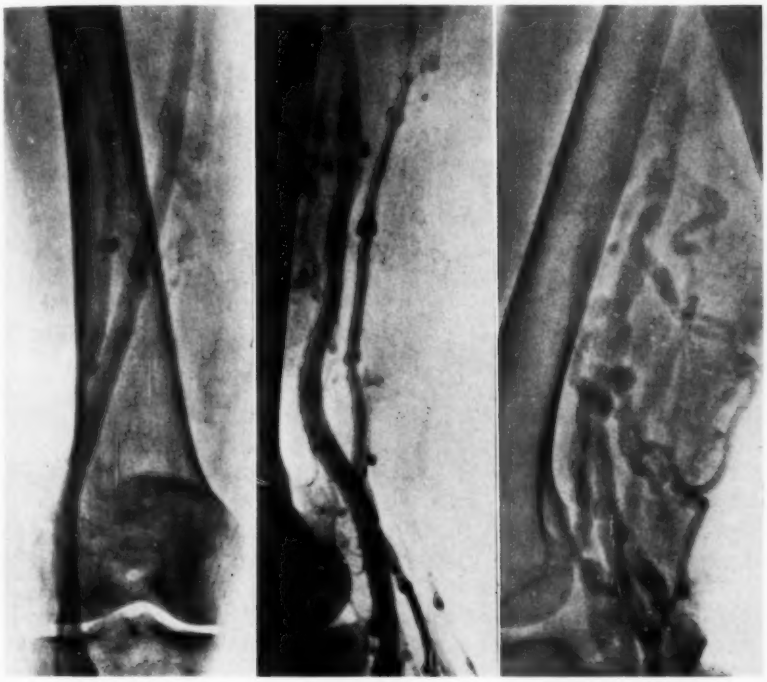
Distribution and localization of changes in the present material

Since the purpose of my investigations has been to explore the morphology of the deep veins I have classified the material according to changes in these veins.

A. *Cases with normal deep veins* (98 extremities). The deep veins were completely normal in 75 cases; in an additional 23 cases the posterior tibial vein was tortuous behind the medial malleolus. As mentioned above, this part of the vein may be considered superficial rather than deep, and when only such a change was present, the deep veins have been considered normal. In a few cases I have observed a deviation from the findings in my normal material (GREITZ 1955), consisting of a direct connection between the short saphenous vein and the dorsal arch of the profunda femoris vein. This has, however, been considered an anomaly (Fig. 9 a, b).

In 15 cases the phlebogram was completely normal, *i. e.* all contrast-filled veins were normal. These patients were relatively young (22 to 44, average 37), and only one had varices. In this case, the contrast medium passed almost exclusively through the deep veins. In the other cases, the roentgen examination had been requested because of chronic or subacute edema.

In all the other cases, superficial varices were present almost without exception. All superficial main trunks have been observed to be varicose; either one at a time or several at the same time. Not unfrequently



a b c

Fig. 9. a, b. Anastomosis between the small saphenous vein and the dorsal arch of the profunda femoris vein, presumably an anomaly. c. Varicose muscular veins in the thigh.

the long saphenous vein was varicose and the short saphenous vein normal; sometimes the opposite occurred. These veins could also have normal appearances, even though there were extensive varices in the areas they drained. In cases with very large varices the long saphenous vein was as a rule tortuous, dilated, and filled as high as the thigh; cases with non-filling of this vein in the upper part of the limb do not as a rule have a great number of varices. Filling of the same vein up to the sapheno-femoral junction was, however, obtained approximately as frequently as in the normal material (GREITZ 1955).

Varicose muscular veins were found in the calf in 26 cases; in only 3 cases, in the thigh.

In 56 cases, there were pathologically changed communicating veins in the lower part of the leg. This is a minimum figure, since a communicating vein may be hidden behind large varices. A communicating

vein just above the medial malleolus was seen to be distended in 22 cases (Fig. 6 b).

Pathologic communicating veins were observed in the thigh in 11 cases, only one of them being laterally situated, while the rest lay mainly in the dorsal or medial region.

B. *Cases with dilated and/or tortuous deep veins* (56 extremities). More or less extensive tortuosity without marked widening was present in 18 cases, while dilatation without or with only very slight tortuosity occurred in 25 cases. In 13 cases distension and tortuosity were combined; in other words, deep varices were present (Fig. 3). VERNEUIL's statement that deep varices are more common than superficial ones is therefore certainly erroneous. Even though one includes varicose changes in all the subfascial veins among the deep varices, such varices are found in phlebography only half as often as superficial varices.

Thirty-eight cases thus had dilated deep veins. In 10 of these, the dilated veins were found only in the lower part of the limb, and in 8 cases the dilatation extended over the entire limb. The popliteal vein was distended in 22 cases, and the femoral vein in 15.

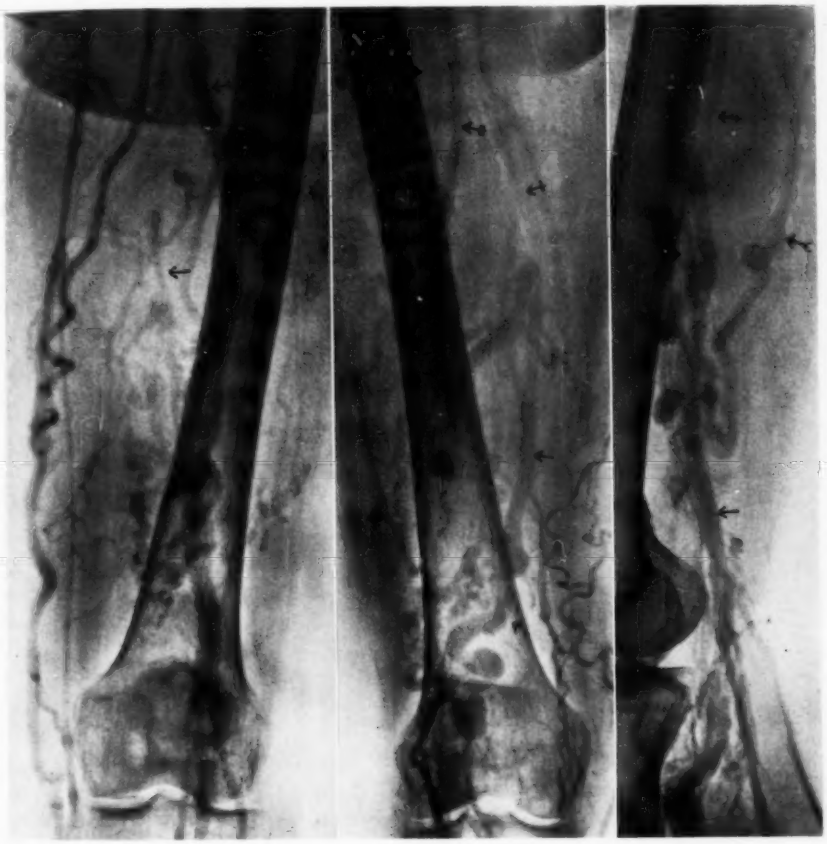
Dilatation of the communicating veins in the lower part of the leg was found in 34 of the 38 cases with distended deep veins, *i. e.* much more often than in patients with normal deep veins. Dilated muscular veins in the calf also seem to be more common (24 out of the 38 cases), but otherwise there was no roentgenologic difference between these patients and those with normal deep veins.

The 23 cases with tortuous but not distended deep veins presented no other differences from those described under A.

C. *Cases with post-thrombotic deep veins* (124 extremities). Out of this material, 53 were right and 71 left legs, about the same proportion as in the entire material.

In cases in which extensive varices are filled, it may be difficult to obtain a clear concept of the deep veins. In 22 out of these 124 cases it has therefore not been possible to arrive at a more definite diagnosis than "most likely post-thrombotic changes".

In all cases there was filling of deep veins, but in 9 cases patency could not be demonstrated. In 3 cases there was no contrast filling of the deep veins in the lower part of the limb; in 6 cases non-filling occurred in a more proximal part, but never in the pelvis. This corresponds to such a high figure for re-canalization (115/124 = 93 %) that one is tempted to believe that non-filling of the deep veins in post-thrombotic extremities is always due to technical reasons. In recent years, several authors have pointed out the high frequency of re-canalization; LINDE (1949) gives the figure as 73 %, HÖJENSGÅRD (1951) 91 % and DOW



a b c

Fig. 10. Post-thrombotic limbs. a. The lower part of the femoral vein is very narrow and irregular (↑), the upper part not filled. Several anastomoses, one of which is seen lateral to the femur, connect the femoral and the profunda femoris veins ( ). b, c. At the site of the popliteal vein a varicose vessel (↑) is seen, probably the dilated medial concomitant vein. The femoral vein is narrow and irregular ( ). The main trunk (↑) and the dorsal arch (↑) of the profunda femoris vein are filled.

(1951) 100 %. But even though one can expect re-canalization in practically all cases, phlebography must still be considered valuable, since the re-canalized veins are often pathologically modified to a high degree.

In the 115 cases with patency through the deep veins, the post-thrombotic changes were in 31 cases situated only in the veins of the lower part of the leg, while in 9 cases these veins were completely normal.

a b c

Fig. 11. Post-thrombotic changes. a, b. Note the main trunk (↑), the dorsal (↑), and the ventral (↕) arch of the profunda femoris vein. c. Same caption as for a, b. Note the varicosity of the dorsal arch.

One case had post-thrombotic changes only in the pelvis. In the remaining 75 cases with patency, *i. e.* the majority, the changes were thus situated in both the lower and the upper part of the extremity. Twenty-three cases had such changes in the veins of the pelvis as have been described by MARTORELL (1943) as post-thrombotic; at the site of the iliac vein there was either an irregular vessel or collateral channels, which he termed periiliac veins; an anastomosis was sometimes formed by a gluteal vein.

BAUER (1942) held that the venous flow in post-thrombotic limbs practically without exception passed through the superficial veins. OLIVIER (1945, 1948) described, however, deep collateral channels,

especially in the thigh, where the profunda femoris vein could assume such a function.

When this is the case, the anastomoses to the perforating veins and sometimes also to the veins in the quadriceps are widened, so that in addition to the main trunk of the profunda femoris both a dorsal and a ventral arch are filled (Figs. 10, 11). This type of collateralization occurred in no less than 22 out of 71 extremities with post-thrombotic changes in or at the site of the femoral vein. Sometimes the femoral vein was not filled; sometimes an irregular and often narrow channel was present in its place (Fig. 10). Cases with this type of deep collateralization have been included in the group of cases with patency of the deep veins.

Fig. 12. Post-thrombotic limb. A great number of small irregular veins are present subfascially.

The filling of the deep veins was often poorer than that of the superficial veins in the post-thrombotic extremities (Fig. 7 c). In 97 of the 120 post-thrombotic limbs which had not previously been operated on, *i. e.* 81 ± 2.1 %, the long saphenous vein was filled up to the sapheno-femoral junction. In my normal material (GREITZ 1955), the corresponding figure was 40 ± 5.1 %, and in cases with varices but normal deep veins 49 ± 3.6 %. The difference is so large that it may be considered most likely that the long saphenous vein, at least in some cases, functions as a collateral. This vein was strikingly often dilated without being markedly tortuous, and even when it had normal appearances its width in the thigh was generally at the upper margin of normal variations, or 5 to 6 mm.

COCKETT (1953) has pointed out that following a thrombosis a large number of superficial and very small tortuous collaterals may often be seen. Such small veins are, however, also found subfascially (Fig. 12). They are seen particularly often in a position corresponding to the distal part of the deep flexor sheath just behind the fibula and the tibia. They

are much more frequent in post-thrombotic limbs (more than 3/4 of the cases) than in limbs with normal deep veins (1/4 of the cases).

D. *Cases with acute thrombosis* (2 extremities). The changes were identical in the two cases (see Fig. 1 and the corresponding case report).

SUMMARY

Two hundred and eighty (280) extremities have been examined by phlebography in the erect position. The technique used has proved suitable for investigation of the deep venous circulation, not only in the lower but also in the upper part of the leg. In most cases, patency of the iliac vein may be demonstrated. Changes in the superficial, muscular, and communicating veins, may be studied both in the calf and in the thigh. The technique does not permit a complete investigation of the entire venous circulation, but it constitutes a good basis which may be supplemented by other examinations, such as pelvic phlebography, retrograde phlebography, varicography, and arteriography.

ZUSAMMENFASSUNG

Mit der Phlebographie in aufrechter Stellung sind 280 Extremitäten untersucht worden. Es hat sich gezeigt, dass die benutzte Technik für die Untersuchung der tiefen venösen Zirkulation geeignet ist, und zwar nicht nur im unteren sondern auch im oberen Abschnitt des Beines. In den meisten Fällen kann die V. iliaca deutlich dargestellt werden. Veränderungen der oberflächlichen, der tiefen und der kommunizierenden Venen können sowohl in der Wade als auch im Oberschenkel studiert werden. Die Technik gestattet keine vollständige Untersuchung der gesammten venösen Zirkulation; sie schafft jedoch eine gute Basis, welche durch andere Untersuchungen wie die Beckenphlebographie, die retrograde Phlebographie, die Varicographie und die Arteriographie vervollständigt werden kann.

RÉSUMÉ

Deux cent quatre-vingts (280) membres inférieurs ont été examinés par phlébographie en position debout. La technique utilisée s'est montrée propre à l'examen de la circulation veineuse profonde, non seulement de la partie inférieure, mais aussi de la partie supérieure du membre inférieur.

Dans la plupart des cas, la perméabilité de la veine iliaque peut être mise en évidence. Les modifications des veines superficielles, musculaires, et communicantes peuvent être étudiées dans le mollet et dans la cuisse. Cette technique ne permet pas un examen complet de la totalité de la circulation veineuse, mais elle constitue une bonne base qui peut être complétée par d'autres examens tels que la phlébographie pelvienne, la phlébographie rétrograde, la varicographie et l'artériographie.

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AUS DEM MAX-PLANCK-INSTITUT FÜR BIOPHYSIK (DIREKTOR: PROF. B. RAJEWSKY), FRANKFURT (MAIN), UND DER PHYSIKALISCH-TECHNISCHEN BUNDESANSTALT, BRAUNSCHWEIG, DEUTSCHLAND

VERIFIZIERUNG DES »r« BEI RÖNTGENSTRAHLEN IM BEREICH 5 KV BIS 50 KV ERZEUGUNGSSPANNUNG[1]

von

E. Bunde, A. Sewkor, B. Rajewsky und *R. Jaeger*

I. Einleitung

Nachdem durch den Krieg die deutschen Standardanlagen für Röntgendosismessung in der Physikalisch-Technischen Reichsanstalt, Berlin, verloren gegangen waren, musste eine neue Möglichkeit für die Verifizierung des internationalen »Röntgen« geschaffen werden. Dies erfolgte auf Veranlassung von B. RAJEWSKY in der Aussenstelle Ockstadt des Max-Planck-Institutes für Biophysik, Frankfurt (Main), wie es kürzlich R. JAEGER und B. RAJEWSKY in der »Strahlentherapie« ausführlich beschrieben haben (17).

Dabei handelte es sich jedoch zunächst nur um eine Messanlage für den Spannungsbereich von 40 bis 300 kV. Es galt nunmehr, auch eine Anlage für die Standardmessung unterhalb 40 kV aufzubauen.

II. Kurzer Überblick über die bisherige Entwicklung der Weichstrahl-Standarddosimetrie

Als Ionisationskammer für definitionsgemässe Absolutmessungen der Röntgenstrahlendosis kommen prinzipiell zwei verschiedene Typen in Betracht: die Zylinderkammer- und die Parallelplattenkammer (s. u. Abschn. IV). In Deutschland wurden für die Standarddosimetrie von Anfang an (1924) Zylinderkammern verwendet, ebenso in Schweden. Sie wurden vor allem von HOLTHUSEN und BECKER (1, 13), BEHNKEN und JAEGER (2, 3, 4), BERG, SCHWERDTFEGER und THALLER (5), KÜSTNER und Mitarbeitern (10, 21, 22), GAERTNER (8) und THORÆUS (34) untersucht. Auch die neue Standardanlage in Ockstadt wurde mit einer Zylinderkammer ausgestattet. Dagegen bevorzugten die angelsächsischen Länder das Prinzip der Parallelplattenkammer, das in USA besonders von TAYLOR und SINGER (29, 30, 31), in England von KAYE und BINKS (18) näher untersucht wurde.

Bei der Red. am 30. VIII. 1954 eingegangen.

[1] Über die Ergebnisse dieser Arbeit berichtete JAEGER auf dem VII. Internat. Radiologenkongress, Kopenhagen 1953.

Die zunächst gebauten Standardkammern waren jeweils für Strahlungen oberhalb etwa 40 kV Erzeugungsspannung gedacht. Sowohl in Deutschland wie in USA wurde im Jahre 1934 auch je eine Standardanlage für Weichstrahldosimetrie (ab 3 kV Erzeugungsspannung) fertiggestellt, in beiden Fällen im wesentlichen verkleinerte Modelle der grossen Standardkammern (JAEGER (15), TAYLOR und STONEBURNER (32). Beide Laboratorien hatten zur Sicherung der Anzeigen der kleinen Standardkammern diese mit ihren grossen Kammern verglichen, und zwar im Bereich zwischen 50 und 150 kV (JAEGER) bzw. 40 und 90 kV (TAYLOR und STONEBURNER) und hatten befriedigende Übereinstimmung gefunden. Allerdings war in beiden Fällen kein direkter Vergleich vorgenommen, sondern eine dritte Kammer, deren Wellenlängenunabhängigkeit mit der grossen Kammer geprüft war, als Übertragungskammer benutzt worden. Diesen Vergleichsmessungen, die jeweils nur zwei verschieden grosse Ausführungen ein und desselben Kammertyps umfassten, waren jedoch im Jahre 1931 bereits internationale Vergleichsmessungen zwischen den grossen Standardkammern in Deutschland, England und den USA vorangegangen. Sie hatten schliesslich ergeben, dass die gegenseitigen Abweichungen dieser Standardanlagen nicht mehr als 1 % betrugen (KAYE und BINKS (19), TAYLOR (33)). So konnte später mit Recht angenommen werden, dass die gesamte Standarddosimetrie im Strahlenbereich zwischen 3 und 150 kV Erzeugungsspannung auf gesicherter Grundlage ruhte.

1932 hatte ausserdem WILHELMY einen ganz anderen Typ einer absolut messenden Ionisationskammer für sehr weiche Röntgenstrahlen angegeben (36, 37): eine Ionisationskammer von solcher Länge, dass die Röntgenstrahlen in ihr *total* absorbiert werden. Mit Hilfe des gesondert zu messenden Absorptionskoeffizienten kann man damit ebenfalls die Dosis in internationalen »Röntgen« aus der gemessenen Elektrizitätsmenge und dem Querschnitt der Messblende bestimmen. Wilhelmys Kammer (Frankfurt/Main) konnte bei 2 m Länge ohne Überdruck bis maximal 12 kV verwendet werden; mit der in Berlin stehenden kleinen Standardkammer wurde sie nicht verglichen. Durch den Krieg ging auch sie verloren.

Nach dem Kriege stellten sich nun grössere Diskrepanzen zwischen den Anzeigen verschiedener Dosimeter der radiologischen Praxis im Weichstrahlgebiet heraus, obwohl ihre Eichung — mittels Sekundärstandards, die den Krieg überstanden hatten — auf die früheren Standardwerte zurückging. Z. B. zeigte eine Grenzstrahlkammer, die in dieser Weise nach dem Krieg in Deutschland geeicht war, bei einer Kontrolleichung in USA (National Bureau of Standards), deren Standardanlagen ja keinerlei Einbussen erlitten hatten, einen Unterschied beider Eichwerte von rd. 50 %. Demgegenüber nahm die Bedeutung der Weichstrahldosimetrie durch das Aufkommen der Röntgenröhren mit Berylliumfenster nach dem Kriege mehr und mehr zu.

Die Situation zu Beginn des Aufbaus unserer Weichstrahl-Standardanlage war noch durch eine weitere bedeutsame Tatsache charakterisiert: Nach einem neuen internationalen Vergleich aller Standardanlagen der Welt (Australien, Deutschland, England, Neuseeland, Schweden, USA), der im Jahre 1950 durch Herrn Dr. Roth, Neuseeland, vorgenommen worden war — allerdings mit einem handelsüblichen Victoreen-Dosimeter als Übertragungsinstrument — schien es so, als ob zwar die Gruppe der Zylinderkammern und die Gruppe der Plattenkammern unter sich gut übereinstimmten, jedoch zwischen beiden Gruppen ein systematischer, mehrere Prozent betragender Unterschied bestünde. (Herr Dr. Roth berichtete darüber auf dem VII. Internationalen Radiologenkongress in Kopenhagen 1953.)

III. Aufgabenstellung

Einerseits auf Grund der Ergebnisse der Roth'schen Vergleichsmessungen, andererseits auf Grund der Überlegungen, die in Abschn. IV näher ausgeführt sind, wurde beschlossen, für den neuen Weichstrahl-

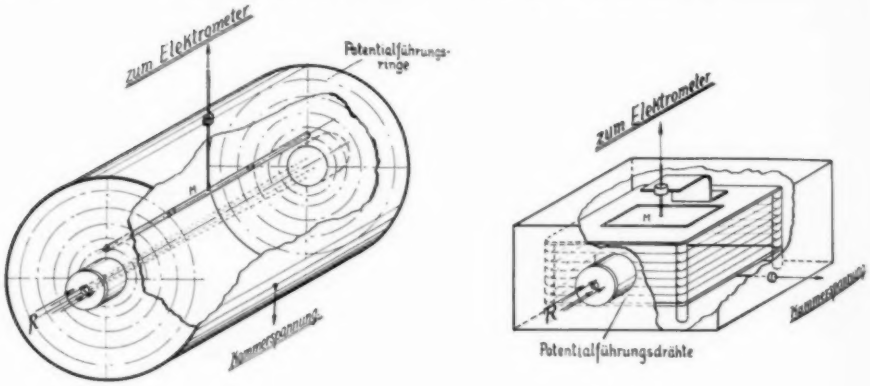
Eichstand in Ockstadt eine Parallelplattenkammer zu bauen. Diese Aufgabe zerfiel in zwei Teile: 1) sollten auf Grund systematischer Messreihen — unabhängig von den Untersuchungen von TAYLOR und STONEBURNER (32) — die optimalen Einzelheiten der endgültigen Kammerkonstruktion sowie besonders die kleinstmöglichen Abmessungen in Strahlrichtung festgestellt werden; 2) sollte das Prinzip der Plattenkammer mit dem Prinzip der Zylinderkammer bezüglich der Verifizierung des »Röntgen« unmittelbar und mit grösstmöglicher Genauigkeit verglichen werden, und zwar in dem Qualitätsbereich, in dem beide Kammern den Anforderungen an eine definitionsgemässe Messung der Dosis in »Röntgen« in gleicher Weise genügen.

Über die Durchführung dieser beiden Aufgaben wird in der vorliegenden Arbeit berichtet. Parallel dazu wurde ausserdem eine neue 2 m lange totalabsorbierende Ionisationskammer nach WILHELMY gebaut und auch ein direkter Vergleich zwischen dieser und der Weichstrahl-Standardkammer (im Überlappungsbereich 5—10 kV) zur Aufgabe gestellt. Darüber ist bereits in einer gesonderten Arbeit berichtet worden (6).

IV. Grundsätzliche Überlegungen

In Abb. 1 sind beide Bauprinzipien einer der Definition des »r« entsprechenden Standardkammer schematisch gegenübergestellt. In beiden Fällen wird die Kammer von einem ausgeblendeten Strahlenbündel (R) durchsetzt, das allseits von soviel Luft umgeben ist, wie es der Reichweite der schnellsten Primärelektronen entspricht, so dass auch alle sekundär gebildeten Ionenpaare gemessen werden. In beiden Fällen wird das Messvolumen seitlich durch die Eintrittsblende, vorn und hinten durch elektrische Feldlinien begrenzt und ist somit durch den Querschnitt der Blende und die effektive Länge der »Messelektrode« (M) bestimmt. Die »Führung« des elektrischen Feldes geschieht dabei erstens durch die vor und hinter der Messelektrode angebrachten geerdeten Schutzelektroden und zweitens durch Potentialführungsringe (Zylinderkammer) bzw. -drähte (Plattenkammer), die über entsprechend viele Hochohmwiderstände miteinander verbunden sind und den Randflächen des eigentlichen Messraumes zwangsweise annähernd denselben Potentialverlauf geben, den sie bei unendlich ausgedehnter Kammer hätten.

Die Unterschiede zwischen beiden Kammertypen bestehen hauptsächlich in folgendem: 1) Bei der Plattenkammer ist die elektrische Feldstärke im ganzen Messraum konstant, während sie in der Zylinderkammer von innen nach aussen mit der Entfernung von der Innenelektrode abnimmt und das Feld ausserdem in Bezug auf das Strahlenbündel unsymmetrisch ist. 2) Die Messelektrode liegt bei der Plattenkammer nicht, wie bei der Zylinderkammer, in dem für die Messung massgebenden Vo-



a. b.

Abb. 1. a) Zylinder-Kammer; b) Platten-Kammer.

lumen, sondern je nach Plattenabstand beliebig weit davon entfernt. Dadurch fällt bei ihr der »Schatteneffekt« der Elektrode weg, und ausserdem kann die Eintrittsblende verhältnissmässig gross gewählt werden, ohne dass die Messelektrode von direkter Strahlung getroffen wird. Letzteres ist besonders für die Absolutmessung sehr geringer Dosisleistungen von Wichtigkeit. Beide Unterschiede sprechen zweifellos zugunsten der Plattenkammer, ebenso wie die Tatsache, dass sie leicht zerlegbar und damit in allen Einzelteilen zugänglich hergestellt werden kann (vgl. Abb. 5).

Die Dosis, die eine Standardkammer bei Messung der Elektrizitätsmenge ΔQ erhalten hat, berechnet sich aus der Formel: $D = \frac{\Delta Q_{[e.s.e.]}}{q_{[cm^2]} \cdot L_{[cm]}} \cdot K\,[r]$[1]

q Blendenquerschnitt; L Länge des Messvolumens. Mit K seien hier alle notwendigen Korrekturen zusammengefasst. Im einzelnen ergibt sich

a) für die Zylinderkammer: $K_{ZK} = K_D \cdot K_S \cdot K_{Sch} \cdot K_{Str} \cdot K_E$

b) für die Plattenkammer: $K_{PK} = K_D \cdot K_S \cdot K_{Sch} \cdot K_{Str}$.

Dabei ist

K_D der Umrechnungsfaktor von der bei der Messung herrschenden Luftdichte auf Normalbedingungen;

K_S ein Korrekturfaktor, der berücksichtigt, dass in der Praxis nie *vollständige* Sättigung vorhanden ist, sondern stets ein kleiner Teil der Ionen rekombiniert;

K_{Sch} der Faktor, der die Schwächung der Strahlung auf dem Weg zwischen Messblende und Mitte der Messelektrode korrigiert;

K_{Str} ein Korrekturfaktor, der berücksichtigt, dass das durchstrahlte Luftvolumen innerhalb der Kammer eine Streustrahlenquelle darstellt und dass durch teilweise

[1] Siehe »Zusatz zur Korrektur«, S. 181.

Absorption dieser Streustrahlung in dem grossen Volumen der Kammer ein Ionenzusatz mitgemessen wird, der nicht der Definition des »r« entspricht. Im Gegensatz zu allen anderen genannten Faktoren ist K_{Str} kleiner als 1.

K_E der Faktor, der den »Schatteneffekt« der Elektrode korrigiert. Dieser Effekt besteht darin, dass die Messelektrode einen Teil der Primärelektronen vorzeitig abfängt, so dass sie nicht in vollem Masse zur Ionisation beitragen.

Werden die Dosismessungen zweier Standardkammern miteinander verglichen, so ist die wichtigste Forderung, dass wirklich in beiden Kammern genau die gleiche Strahlenmenge zur Messung kommt. Das ist das gleiche Problem, das auch bei der Eichung eines beliebigen Dosimeters besteht, und es erwächst vor allem aus drei Faktoren: 1) aus der zeitlichen Inkonstanz der Röntgenapparatur (Spannungs- und Stromschwankungen); 2) aus der Beschaffenheit des Röhrenbrennflecks, seiner Grösse und Struktur sowie seiner eventuellen räumlichen Inkonstanz (Bei Therapieröhren mit Glaswänden ist es nichts Ungewöhnliches, wenn der Brennfleck auf der Anode etwas hin und her wandert); und 3) aus der Notwendigkeit, dass für beide zu vergleichenden Kammern der Abstand Fokus—Messort genau gleich sein muss.

Die erstgenannte Schwierigkeit wird am besten durch Verwendung eines »Monitorgerätes« beseitigt.

Es besteht aus einer mit dünnen, leichtatomigen Folien abgeschlossenen Durchstrahlkammer, die im allgemeinen zwischen Röhre und Eintrittsblende der Standard- bzw. der zu eichenden Kammer in den Strahlengang gestellt wird, und einer Messvorrichtung zur Bestimmung der in der Kammer gebildeten Elektrizitätsmenge in relativen Einheiten. Selbstverständlich muss auch hier für Sättigungsspannung an den Elektroden und für Unabhängigkeit von den durch die Schwankungen der Apparatur bedingten Qualitätsschwankungen gesorgt werden. Um letzteres auch bei weichen Strahlen zu erreichen, muss man die »Monitorkammer« möglichst dicht vor die Messblende setzen; denn die Schwankungen der Durchlässigkeit der Luftschicht zwischen Monitorkammer und Messblende wirken sich auf das Ladungsverhältnis zwischen Standardkammer und Monitorkammer voll aus. Ein geringer Abstand zwischen Monitorkammer und Messblende hat noch den weiteren Vorteil, dass dem Strahlenbündel, das in der Monitorkammer erfasst wird, durch geeignete Einblendung fast der gleiche Querschnitt gegeben werden kann wie dem Strahlenbündel, das in der Standardkammer zur Messung gelangt, so dass eventuelle Brennfleckverschiebungen sich in beiden Kammern in annähernd gleicher Weise auswirken und dadurch praktisch keinen Einfluss auf die Vergleichsmessung haben. Es muss jedoch darauf geachtet werden, dass nicht durch Rückstreuung an den Messblenden Zusatzstrahlung in die Monitorkammer gelangt, die für die zu vergleichenden Kammern etwa verschieden ist. Damit auch eine etwaige Nichtlinearität der Monitoranzeige keine Rolle spielt, werden beide zu vergleichenden Kammern stets bis zur Erreichung ungefähr derselben Anzahl Skalenteile am Monitorgerät bestrahlt.

Als Ergebnis der Messung erhält man auf diese Weise eine auf gleiche Monitoranzeige bezogene Dosis D bzw. Elektrizitätsmenge ΔQ. Die zweitgenannte Schwierigkeit ist umso grösser, je grösser die Brennfleckfläche und je weniger gleichmässig sie mit Elektronen belegt ist.

Abb. 2. Lochkammeraufnahme des Brennflecks einer Weichstrahlröhre mit Berylliumfenster (zweifach vergrössert).

Ein Beispiel grosser Inhomogenität zeigt Abb. 2. Es ist daher notwendig, den Einfluss des Brennflecks bei jeder Standardanlage gesondert zu untersuchen und durch entsprechend sorgfältige Einblendung des Strahlenbündels (mehrere Blenden, keine darf kleiner sein als der Brennfleck; darüber sind noch besondere Untersuchungen durchgeführt worden, über die später berichtet wird) sowie möglichst grossen Fokusabstand der Standardkammer auf ein Mindestmass zu reduzieren.

Der unter 3) genannte Einfluss der Abstandsbestimmung fällt umso mehr ins Gewicht, je näher der Messort am Fokus liegt. Auch dieser Faktor erfordert also möglichst grosse Fokusabstände.

Bei prinzipieller Gleichheit beider Kammertypen muss bei einem Vergleich das Verhältnis der beiden Dosisgrössen $\bar{D}$

$$\frac{\bar{D}_{PK}}{\bar{D}_{ZK}} = \frac{(\overline{\Delta Q})_{PK} \cdot (K_{Sch})_{PK} \cdot (K_{Str})_{PK} \cdot (K_D)_{PK} \cdot (K_S)_{PK} \cdot q_{ZK} \cdot L_{ZK}}{(\overline{\Delta Q})_{ZK} \cdot (K_{Sch})_{ZK} \cdot (K_{Str})_{ZK} \cdot (K_D)_{ZK} \cdot (K_S)_{ZK} \cdot K_E \cdot q_{PK} \cdot L_{PK}}$$

gleich 1 sein bzw. entsprechend der Messgenauigkeit um den Mittelwert 1 herum streuen. Um diese Streuungen so klein wie möglich zu machen und dadurch zu verhindern, dass ein eventuell vorhandener prinzipieller Unterschied der Messysteme durch ungünstiges Zusammenwirken mehrerer Fehler verdeckt wird, haben wir die Messgenauigkeit so weit wie irgend möglich gesteigert. Der grösste Korrekturfaktor K_D hebt sich heraus, wenn beide Messungen unmittelbar hintereinander, also bei gleicher Luftdichte, ausgeführt werden. Auch K_S fällt heraus, wenn die Feldstärke im Messvolumen in beiden Kammern gleich gross ist.

Die Bestimmungsgrössen q sowie die Korrekturfaktoren K_{Sch} konnten durch folgende Messanordnung eliminiert werden, mit der ausserdem der Vorteil verbunden war, dass keinerlei Abstandsmessung in das Ergebnis einging: Die Messblende für die Standardkammern wurde völlig getrennt vom übrigen Kammergehäuse in den Strahlengang gestellt und für beide Kammern ein und dieselbe Blende verwendet; Monitorkammer und Messblende blieben also während der ganzen Vergleichsmessung am selben Ort stehen. Die beiden Kammergehäuse mit den zu vergleichenden Ladungsmessystemen wurden nacheinander so in den Strahlengang geschoben, dass der Abstand Messort—Mitte der Messelektrode in beiden Fällen der gleiche war (vgl. Abb. 7).

Abgesehen von den Korrekturfaktoren K_{Str} und K_E war damit das

Dosisverhältnis nur noch von den beiden Elektrodenlängen und den Grössen ΔQ, deren Bestimmung die eigentliche Messung ausmachte, abhängig:

$$\frac{D_{PK}}{D_{ZK}} = \frac{(\Delta Q)_{PK} \cdot L_{ZK}}{(\Delta Q)_{ZK} \cdot L_{PK}} \cdot \frac{(K_{Str})_{PK}}{(K_{Str})_{ZK} \cdot K_E}.$$

Die Faktoren K_{Str} konnten deshalb nicht ausgeschaltet werden, weil unsere Plattenkammer als Weichstrahl-Standardkammer von vornherein kleinere Abmessungen hatte als unsere Zylinderkammer, deren Durchmesser 40 cm beträgt und deren Messelektrode 29 cm lang ist. Massgebend für den Fehler, den der Streuzusatz hineinbringt, ist aber das Gesamtvolumen der Kammer (siehe S. 178 und 179).

V. Durchführung der Messungen und Ergebnisse

1. Festlegung der konstruktiven Einzelheiten der Weichstrahl-Standardkammer. Praktisch gingen wir folgendermassen vor: Wir bauten zunächst — in enger Anlehnung an das von TAYLOR und STONEBURNER gegebene Vorbild (32) — eine Plattenkammer mit in Strahlrichtung verschiebbarem Messystem (zur direkten Messung des Faktors K_{Sch} in jedem Einzelfall), und zwar so, dass verhältnismässig leicht verschiedene Veränderungen daran vorgenommen werden konnten. Der Einfluss dieser Veränderungen wurde durch Vergleich mit der Zylinderkammer bei 50 kV im einzelnen untersucht. Auf Grund der Ergebnisse und der bei den Messungen gewonnenen Erfahrungen bauten wir dann eine zweite Plattenkammer als Weichstrahl-Standardkammer und verglichen ihre Anzeige mit der der Zylinderkammer mit höchstmöglicher Präzision.

Die charakteristischen Grössen einer Plattenkammer, die für eine Variation in Frage kommen, sind die folgenden: a) Abstand der Platten; b) Breite der Messelektrode; c) Breite der Luftspalte zwischen Messelektrode und Schutzplatte; d) Breite der Schutzelektrode; e) Länge der Schutzelektrode; f) Abstand der Drähte für die Potentialsteuerung.

a) Im Hinblick auf die Höhe der Sättigungsspannung sollte der Plattenabstand nicht grösser sein, als notwendig ist, damit die Weichstrahlkammer noch bis 50 kV die Dosis absolut in »Röntgen« messen kann. Wir wählten 50 mm als fest gegebenen Abstand.

Nach LEA (24) beträgt die *maximale* Reichweite der Photoelektronen bei *monochromatischer* 50-kV-Strahlung zwar 3.3 cm, ihr Anteil an der Gesamtzahl der ausgelösten Elektronen jedoch nur 20 %. Für heterogene Strahlung mit normaler Filterung, wie wir sie verwenden, ist die effektive Wellenlänge etwa doppelt so gross anzusetzen wie die Grenzwellenlänge. Elektronen mit 25 keV Energie haben aber nur noch eine maximale Reichweite von rd. 1 cm. In einer Kammer mit 5 cm Plattenabstand stehen den Elektronen bei enger Ausblendung des primären Strahlenbündels (z. B. 4 mm Blen-

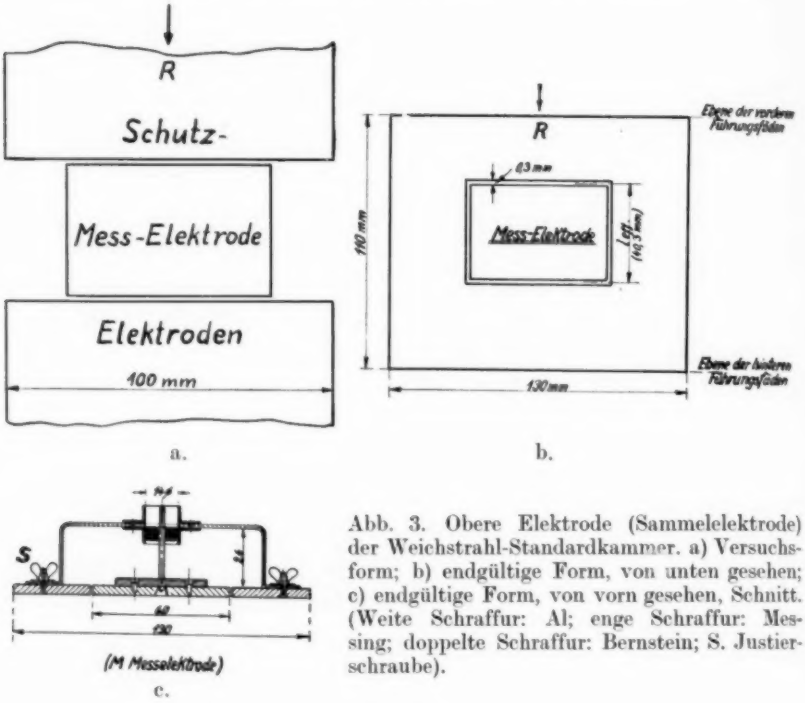


Abb. 3. Obere Elektrode (Sammelelektrode) der Weichstrahl-Standardkammer. a) Versuchsform; b) endgültige Form, von unten gesehen; c) endgültige Form, von vorn gesehen, Schnitt. (Weite Schraffur: Al; enge Schraffur: Messing; doppelte Schraffur: Bernstein; S. Justierschraube).

dendurchmesser) mindestens 2.3 cm zur Verfügung, bei schräg zur Achse des Strahlenbündels verlaufender Bahn entsprechend mehr. TAYLOR und STONEBURNER fanden bei 5 cm Plattenabstand experimentell Wellenlängenunabhängigkeit (innerhalb 1 %) sogar bis 70 kV Erzeugungsspannung (vgl. auch Tab. 5).

Bei den verwendeten Dosisleistungen — bis 10 r/min. — erwies sich bereits eine Elektrodenspannung von 500 Volt als zur Sättigung ausreichend, eine Steigerung der Spannung auf 1,600 Volt brachte keinen messbaren Ladungszusatz (Streuung der Messwerte nur wenige Promille). Wir benutzten stets eine Kammerspannung von 1,000 V = 200 V/cm Feldstärke.

b) Der Einfluss der Breite der Messelektrode wurde zusammen mit dem Einfluss der Breite der Schutzelektrode (d) an einer 10 cm langen Messelektrode untersucht. Die Röhrenspannung betrug dabei 52 kV. Tab. 1 zeigt, dass bereits bei 40 mm Elektrodenbreite das Ladungsdefizit weniger als 1/2 % beträgt. Dabei war es unwesentlich, ob die Breite der umgebenden Schutzplatte gross oder klein war, oder ob seitlich überhaupt keine Erdplatte vorhanden war (Elektrodenform Abb. 3 a). Im Interesse

einfacherer Herstellung und Handhabung und grösserer Zuverlässigkeit entschieden wir uns aber bei der endgültigen Konstruktion der Sammelelektrode für die Form der Abb. 3 b, sicherheitshalber mit einer Elektrodenbreite von 60 mm.

Tabelle 1

Einfluss der Breite der Messelektrode auf die gemessene Ladungsmenge

Röhrenspannung 52 kV; HWS 1.6 mm Al (gemessen mit Zylinder-Standardkammer im Messabstand); Messblende 4 mm ∅; Abstand Fokus—Messblende 1 m; Länge der Messelektrode 10 cm

Elektrodenbreite mm	Gemessene Ladung %
20	94.5
40	99.6
50	99.8
60	100.0
70	100.0

c) Die Breite der Luftspalte zwischen Mess- und Schutzelektrode variierten wir bei einer 4 cm langen Messelektrode von 0.3 bis 2.5 mm. Rechnet man als »effektive Elektrodenlänge« (L_{eff}), wie üblich, die Strecke von der Mitte des vorderen bis zur Mitte des hinteren Luftspaltes, so bedeutet das eine Änderung des Spaltanteils an L_{eff} von 0.75 bis 6 %. Dabei betrug die Länge der Schutzelektrode nach vorn und nach hinten je 10 cm. Wie Tab. 2 zeigt, ist die Spaltbreite bis zu 4 % ohne messbaren Einfluss auf die Ladungsmessung. Dasselbe Ergebnis fand seinerzeit auch JAEGER bei einer analogen Untersuchung an der Weichstrahl-Zylinder-

Tabelle 2

Einfluss der Spaltbreite zwischen Mess- und Schutzelektrode auf die Ladungsmessung

Messbedingungen wie bei Tab. 1, jedoch Länge der Messelektrode 4 cm

Spaltbreite mm	Spaltbreite %	L_{eff} mm	$\Delta C/L_{eff}$ Messwert	$\Delta C/L_{eff}$ Mittel	Bemerkungen
0.3	0.75	40.3	1.328		
			1.331	1.331	Besondere Elektrode m. veränderlicher Spaltbr.
			1.333		
			1.331	1.331	Kontrolle z. Schluss m. ders. Elektr.
			1.331		
			1.333	1.331	Neue Elektrode m. d. gleich. Abmessungen
			1.329		
1.8	4.3	41.8	1.332		
			1.327	1.331	
			1.333		
2.5	5.9	42.5	1.337		
			1.341	1.338	
			1.337		

ΔC bedeutet die für die Ladungs-Kompensation notwendige Kapazitätsänderung bei konstanter Spannung am Kondensator.

kammer der PTR (15). Es bedeutet, dass auch bei einer effektiven Elektrodenlänge von nur 10 mm die Spalte noch 0.4 mm breit sein dürfen. Wir gaben sämtlichen Elektroden der neuen Kammer eine Spaltbreite von 0.3 mm.

d) Siehe b).

e) Damit die Korrektur für die Schwächung der Strahlung innerhalb der Kammer nicht zu grosse Werte annimmt und damit sich die Qualität der zu messenden Strahlung nicht merklich innerhalb der Kammer ändert, muss die Luftstrecke, die das Strahlenbündel in der Kammer durchsetzt, ehe es zur Messung gelangt, so klein wie möglich gemacht werden. Es war daher vor allem zu klären, wie weit die Schutzelektrode die Messelektrode nach *vorn* mindestens verlängern muss, damit Veränderungen an der vorderen Wand des geerdeten Kammergehäuses (z. B. Grösse der Öffnung zum Eintritt der Strahlen, Abstand von den vorderen Potentialführungsdrähten) keinen Einfluss auf die zur Messelektrode gelangende Ionenmenge haben, mit anderen Worten: damit die Gewähr gegeben ist, dass die endliche Ausdehnung der Kammer bzw. des elektrischen Feldes nach vorn und hinten keine Verzerrung der das Messvolumen durchsetzenden Kraftlinien mit sich bringt und somit dessen Länge auch wirklich mit der gemessenen effektiven Elektrodenlänge übereinstimmt.

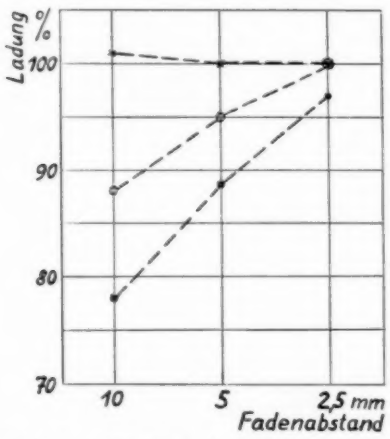


Abb. 4. Einfluss des Abstandes der Potentialführungsfäden auf die Ladungsmessung. × ohne Vorderwand; ● geerdete Vorderwand in 2 mm Abstand vor den vorderen Fäden; ○ geerdete Vorderwand in 13 mm Abstand vor den vorderen Fäden. Messbedingungen wie bei Tab. 2, jedoch Länge der Messelektrode 2 cm.

Diese Frage wurde in Verbindung mit Punkt f) untersucht.

f) Bei unserer Versuchskammer betrug der Abstand der Potentialführungsdrähte zunächst 10 mm und die Länge der vorderen und ebenso die der hinteren Schutzelektrode 10 cm. Dabei war keinerlei Einfluss der Vorderwand auf das Messergebnis festzustellen. Da TAYLOR angegeben hatte (33), dass — bei einem Abstand der Drähte von 6 mm — die vordere Schutzelektrode nicht länger als 2 cm zu sein brauchte, wahrscheinlich aber noch kürzer sein könnte, nahmen wir dann eine Sammelelektrode, bei der die vordere Schutzplatte nur 1 cm lang war, und variierten den Abstand der Führungsdrähte. Dabei gingen wir von den zunächst benutzten Aluminiumdrähten ab und verwendeten stattdes-

sen dünne, durch Graphitüberzug leitend gemachte Perlonfäden, die sich sehr gut spannen lassen (s. Abb. 5). Abb. 4 gibt schematisch die Ergebnisse. Auch bei 2.5 mm Fadenabstand blieb zwischen den Messungen mit und ohne (geerdete) Vorderwand noch ein Unterschied von 3 %, wenn die Vorderwand bis auf 2 mm an die vorderen Fäden herangebracht wurde. Erst wenn sie 13 mm entfernt blieb, ergab sich kein messbarer Einfluss mehr. Eine geerdete Wand musste demnach von der Vorderfläche des Messvolumens mindestens eine Distanz von 23 mm haben. Kleinere Abstände zwischen den Potentialführungsfäden als 2.5 mm hielten wir nicht für zweckmässig, da bei den Messungen an sehr weichen Strahlen die Schwächung durch die vorderen Fäden schon eine Rolle spielt (bei 8 kV in 60 cm Fokus-Abstand rd. 2 %). So setzten wir die Versuche mit je einer 20 und 30 mm langen vorderen Schutzplatte fort. Tab. 3 zeigt, dass erst bei 30 mm der Einfluss der vorderen Kammerwand geringer ist als die relative Ungenauigkeit der Messungen. In diesem Punkt ergab sich also keine vollständige Übereinstimmung mit den amerikanischen Angaben.

Tabelle 3

Einfluss der Länge der vorderen Schutzelektrode auf die Ladungsmessung

Messbedingungen wie bei Tab. 2

Abstand Fadenebene —Messelektrode mm	Abstand Vorderwand —Fadenebene mm	$\overline{\Delta C}$ Messwert pF/Skt	$\overline{\Delta C}$ Mittel pF/Skt
20	6	5.31	
		5.27	5.28
		5.29	
		5.27	
	15	5.32	
		5.34	5.32
		5.31	
30	6	5.30	
		5.34	5.31
		5.29	
	15	5.36	
		5.29	5.32
		5.31	
	ohne Vorderwand	5.32	
		5.35	5.32
		5.31	

Die endgültige Form für die Sammelelektrode unserer Standardkammer ist in Abb. 3 b und c gezeichnet. Die Elektrode ist aus einer 3 mm starken Aluminiumplatte hergestellt, die an der unteren Seite graphitiert ist. Die Messelektrode ist durch Luft, die Zuführung zum Elektrometer durch

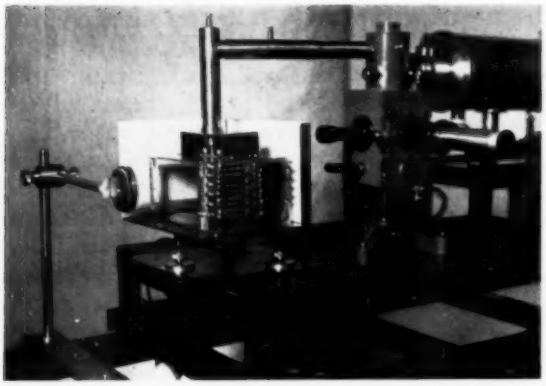


Abb. 5. Ansicht der Weichstrahl-Standardkammer des M. P. I. f. Biophysik, mit freistehender Eintrittsblende. Die Vorderwand, eine Seitenwand und der Deckel der Kammer sind abgenommen.

Bernstein gegen die Schutzelektrode isoliert. Die genaue Einjustierung der Messelektrode in die Ebene der Schutzelektrode, auf die es umso mehr ankommt, je kürzer die Messelektrode ist (Einfluss etwaiger Feldverzerrung), erfolgt mittels der Justierschrauben S und Zwischenlegung dünner Folien (Abb. 3 c). Die ganze Platte wird mit 4 Schrauben auf Trolitulsäulen, die die Potentialführungsfäden und die spannungsführende untere Platte halten, festgeschraubt und ist auf diese Weise leicht auswechselbar. Das gewünschte Potential erhalten die Fäden durch 20 Festwiderstände zu je 25 MOhm, über die die Kammerspannung gleichmässig abfällt. Sie sind seitlich an einer Plexiglaswand im Innern des geerdeten Kammergehäuses angebracht. Abb. 5 zeigt die Ansicht der geöffneten Kammer.

Die gesamte Messanordnung am Weichstrahl-Eichstand ist auf Abb. 6 zu sehen. Der Zentralstrahl verläuft horizontal. Ionisationskammer, Elektrometer und Kompensationskondensator sind gemeinsam auf einem Wagen montiert, der auf einer 5 m langen Schienenbahn beliebig zu verschieben ist. Jedoch ist die Eintrittsblende nicht mit der übrigen Kammer verbunden, sondern für sich allein auf einer ebenfalls frei verschiebbaren Querschiene befestigt. Dadurch konnte das Messystem der Kammer starr mit Elektrometer und Kondensator verbunden werden (Isolation nur durch Luft und Bernstein), und es konnten trotzdem Luftschwächungsmessungen auch über grössere Längen in einfacher Weise durchgeführt werden. (Eine ähnliche Anordnung ist auch in der unter 7) zitierten Arbeit benutzt worden). Die Messblende wird in ihre Halterung so eingeschraubt, dass die Ebene der das Strahlenbündel begrenzenden Öffnung (der »Messort« der Kammer) von der Hinterfläche der Halterung bei allen Blenden denselben Abstand hat (8.4 mm), so dass ihre Auswechslung den Fokusabstand des Messortes nicht beeinflusst (vgl. Abb. 5). Die Blenden, deren Durchmesser von 1 bis 20 mm variiert, bestehen aus 2 mm starken Hartblei-Scheiben, die vorn und hinten mit Messing abgedeckt und konisch

ausgedreht sind, wie es von KÖNIG angegeben wurde (20); vgl. auch (17). Der Messort befindet sich an der Grenzfläche zwischen Messing und Blei. Auf der Hinterwand des Kammergehäuses trifft das Strahlenbündel auf eine 5 mm dicke Bleischeibe, die — nach RAJEWSKY — mit einer Kupfer- und einer graphitierten Aluminiumscheibe von je 1 mm Dicke belegt ist. Die erste Blende zur Eingrenzung des Strahlenbündels ist unmittelbar an der Röhre angebracht, die zweite Blende vor der Monitorkammer. Eine dritte Blende wird noch zwischen Monitorkammer und Messblende eingeschaltet zur Vermeidung von Rückstreuung der Messblende in die Monitorkammer, — sie ist in Abb. 6 u. 7 der Übersichtlichkeit wegen weggelassen worden. Voltmeter und Potentiometer sind auf einem zweiten Wagen auf der Schienenbahn angeordnet.

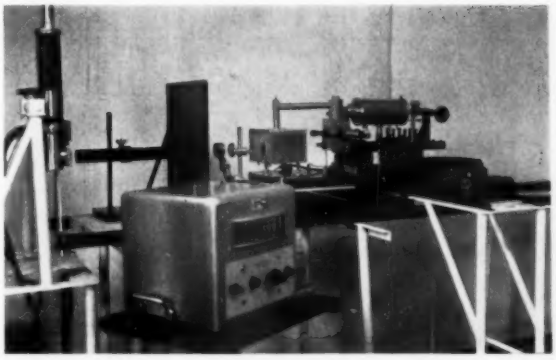


Abb. 6. Ansicht der Messanordnung am Weichstrahl-Eichstand. (Bei der Aufnahme wurde jedoch die Kammer näher an die Messblende herangeschoben.) Im Vordergrund: das Monitorgerät.

2. Exakter Vergleich zwischen der Weichstrahl-Plattenkammer und der Hartstrahl-Zylinderkammer bei 30 und 50 kV Erzeugungsspannung. Der *direkte* Vergleich der beiden Standardkammern liess sich in einfacher Weise vornehmen; denn bei unseren beiden Eichständen wurden die Schienenbahnen mit gleicher Spurweite gebaut und der Zentralstrahl in gleicher Höhe über der Mitte der Bahn einjustiert. Daher können beide Messwagen und alle Querschienen wahlweise an dem einen oder dem anderen Eichstand verwendet werden. Die bleigepanzerte grosse Zylinderkammer wird dabei mit Hilfe einer 1.50 m langen fahrbaren Schienenbahn transportiert.

Die endgültigen Vergleichsmessungen erfolgten bei 52 kV, 1.6 mm Al HWS (gemessen mit der Hartstrahl-Standardkammer im Messabstand), am Hartstrahl-Eichstand mit einer Siemens-Doglas-Tiefentherapieröhre, die an einem »Stabilivolt«-Apparat (Greinacherschaltung) betrieben wurde, und bei 30 kV, 0.4 mm Al HWS (gemessen mit der Weichstrahl-Standardkammer im Messabstand), am Weichstrahl-Eichstand mit einer Machlett-Weichstrahlröhre AEG 50 mit Molybdänanode und Berylliumfenster, betrieben an einem im Institut gebauten »Proton«-Apparat nach Dessauer (ebenfalls Greinacherschaltung). (Für die Freund-

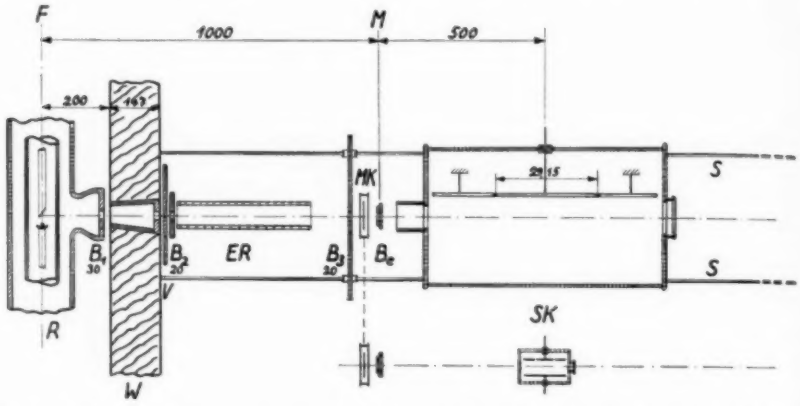


Abb. 7. Schema der Messanordnung am Hartstrahl-Eichstand beim Vergleich der beiden Standardkammern. Masse in mm. F Fokus; M Messort; R Röntgenröhre mit Tutohaube; W Wand, die den Messraum vom Hochspannungsraum trennt; V Verschlussklappe; SK Standardkammer; S—S doppelte Schienenbahn; MK Monitorkammer; B Blende (B_1 verschiebbarer Blendenwagen, B_e Eintrittsblende für die Standardkammer. Die Zahlen geben die Blendendurchmesser an); ER Eisenrohr zur Abschirmung der Streustrahlung aus der durchstrahlten Luft.

lichkeit der leihweisen Überlassung dieser Röhre sei der Fa. Rich. Seifert, Hamburg, herzlich gedankt.) Die beiden Messanordnungen zeigen Abb. 6 und 7.

Die von der Messelektrode gesammelte Elektrizitätsmenge wurde nach einer Kompensationsmethode gemessen, teils nach Townsend: $\Delta Q = C \cdot \Delta U$, teils nach Hartshorn: $\Delta Q = U \cdot \Delta C$ (U Spannung, C Kapazität, das Zeichen Δ gibt den Hinweis auf die bei der Ladungsmessung variierte Grösse, vgl. (16)). Als Nullinstrument diente ein Wulf'sches Einfadenelektrometer (eingestellte Empfindlichkeit etwa 0.02 Volt/Skt), als Kompensationskondensator ein Kapazitätsvariator nach Harms-Gerdien bis 570 pF. Die Kompensationsspannung wurde einer 100-Volt-Akkumulatorenbatterie entnommen, an einem Spezial-Drahtpotentiometer (10 kOhm) abgegriffen und mit einem Präzisionsvoltmeter mit 4 Messbereichen: 3, 15, 30 und 150 Volt gemessen. Bei beiden Kammern wurde *derselbe* Kondensator und *dasselbe* Voltmeter benutzt, so dass die systematischen Fehler beider Geräte das Dosisverhältnis nicht beeinflussen konnten. Auch die Grösse der Kapazität wurde beim Wechseln der Kammern beibehalten, der Kondensator also überhaupt nicht verstellt; nur beim Voltmeter musste man den Messbereich verändern, um den Unterschied der Messvolumina in beiden Kammern auszugleichen. Dieser war, da die Eintrittsblende in beiden Fällen dieselbe war, nur durch die

verschiedenen Längen der Messelektroden gegeben: 4.03 cm bei der Weichstrahlkammer gegenüber 29.15 cm bei der Hartstrahlkammer.

Zur Ladungsmessung an der Monitorkammer wurde ein »Kondiometer« der Physikalisch-Technischen Werkstätten, Freiburg/Br., benutzt, das dieser Aufgabe durch besondere Schaltung und eine Spezialverbindung zwischen Messgerät und Durchstrahlkammer angepasst wurde. Die Messmethode war dabei im Prinzip dieselbe wie an der Standardkammer (Kompensation nach Townsend, Ablesung jedoch nur in relativen Einheiten). Die Monitorkammer wurde, ebenso wie die Standardkammer, in der Werkstatt des M. P. I. angefertigt. Ihr Volumen kann durch Herein- und Herausschrauben der beiden äusseren Membranen (graphitierte, straff gespannte Polyäthylenfolien von 0.02 mm Dicke) in weiten Grenzen geändert werden; im übrigen erfolgt die Anpassung der Messempfindlichkeit an die Dosisleistung wie bei der Standardkammer durch geeignete Wahl der Kammerblende und der Kompensationskapazität sowie der Kompensationsspannung.

Die übrigen Messbedingungen gehen aus Tab. 4 hervor, in der die Ergebnisse der endgültigen Vergleichsmessungen zusammengestellt sind. Alle Messungen erfolgten völlig getrennt voneinander, an verschiedenen Tagen, teilweise auch mit verschiedenen Beobachtern.

Tabelle 4

Ergebnisse der endgültigen Vergleichsmessungen an den beiden Standardkammern

Röhrenspannung kV	Röhrenstrom mA	Gesamtfilter mm Al	HWS mm Al	FA cm	Blendendurchm. mm	Messmethode u. Kompensationskapaz.	$\bar{D}_{PK}$[1] / D_{ZK}
30.5	15	0.5	0.4	60	2.99	ΔU; 302 pF	0.991
		+ 1 Be			4.07	ΔU; 250 pF	0.990
						ΔU; 337 pF	0.994
						ΔC	0.994
52	12	2.7	1.6	100	4.07	ΔU; 138 pF	0.992
					5.57		0.993
					4.07		0.995
							0.993

[1] $\bar{D}$ Dosis bezogen auf einen bestimmten Ausschlag am Monitorgerät.

MW = 0.993 ± 0.001

Die Messwerte der letzten Spalte wurden folgendermassen gewonnen: Durch Benutzung desselben Kondensators mit fest eingestelltem Kapazitätswert für beide zu vergleichenden Ladungsmessungen war $C_{PK} = C_{ZK}$, und die auf S. 169 angegebene Formel für das Dosisverhältnis reduzierte sich auf die Form

$$\frac{\bar{D}_{PK}}{D_{ZK}} = \frac{(\Delta U)_{PK} \cdot L_{ZK}}{(\Delta U)_{ZK} \cdot L_{PK}} \cdot K \qquad K = \frac{(K_{Str})_{PK}}{(K_{Str})_{ZK} \cdot K_E}$$

Die Schwächung durch die vorderen Potentialführungsfäden der Plattenkammer betrug in dem verwendeten Wellenlängenbereich weniger als 1 ‰ und konnte daher vernachlässigt werden.

Die Elektrodenlängen L sind vor der Messung bekannt, die ΔU-Werte wurden unmittelbar am Voltmeter abgelesen und durch die am Monitorgerät abgelesene Anzahl Skalenteile dividiert. Den Korrekturfaktor K setzten wir mit 1.007 ein, wobei wir uns auf Messungen von K_{Str} stützten, die KÜSTNER an Zylinderkammern verschiedenen Durchmessers und bei verschiedenen Strahlenqualitäten gemacht hat (22). Leider variierte er allerdings diese nur bis herab zu 60 kV, so dass wir für den beim Vergleich verwendeten Spannungsbereich zwischen 30 und 50 kV auf Extrapolation angewiesen waren.

Es ergibt sich dann für eine Zylinderkammer von 70 cm ∅ (mit einer Länge der Messelektrode von 28 cm) bei Extrapolation auf 40 kV: $(K_{Str})_{70\ cm} = 0.986$. Das Verhältnis des Streuzusatzes bei 40 cm Kammerdurchmesser zu dem bei 70 cm ∅ beträgt nach seinen Angaben (interpoliert) $\frac{26}{43} = 0.6$, so dass man für $(K_{Str})_{40\ cm} = (K_{Str})_{ZK} = 1.000 - 0.014 \cdot 0.6 = 0.992$ erhält. Unsere Plattenkammer hat nicht nur einen wesentlich kleineren Durchmesser, sondern auch eine rd. 7 mal kleinere Elektrodenlänge. Für diese Abmessungen lässt sich aus den von KÜSTNER angegebenen Werten eine rd. 50 mal kleinere Korrektur, d. h. ein Wert für $(K_{Str})_{PK} = 1.000$ extrapolieren. K_E betrug in diesem Falle — nach WEHRHEIM (35) — 1.001. Daraus folgt, dass K = 1.007 ist.

3. Messung der Wellenlängenabhängigkeit der Weichstrahl-Standardkammer bis 180 kV am Hartstrahl-Eichstand. Schliesslich wollten wir uns noch einen Überblick verschaffen, wie sich die neue Weichstrahl-Standardkammer bei höheren Röhrenspannungen verhält, und schlossen sie bis hinauf zu 180 kV an die grosse Standardkammer an. Das Ergebnis zeigt Tab. 5. Bei 70 kV beträgt das Defizit der in der Weichstrahlkammer gemessenen Ladung erst 1.4 %, in befriedigender Übereinstimmung mit den Ergebnissen von TAYLOR und STONEBURNER (vgl. S. 170).

Tabelle 5

Wellenlängenabhängigkeit der Weichstrahl-Standardkammer

Röhrensp. kV	Ges. filter mm	HWS mm	Ladungsmessung %
30	1 Be + 0.5 Al	0.45 Al	100.0
52	2.7 Al[1]	1.58 Al	100.0
70	0.2 Cu + 0.5 Al	0.10 Cu	98.6
90	0.2 Cu + 2 Al	0.19 Cu	95.7
120	0.35 Cu + 1 Al	0.46 Cu	91.7
150	0.65 Cu + 1 Al	0.85 Cu	90.3
180	0.85 Cu + 1 Al	1.28 Cu	90.5

[1] Eigenfilterwert der Doglasröhre; 0.15 mm Cu od. 2.7 mm Al.

VI. Diskussion der Messgenauigkeit und Schlussfolgerungen

Der maximale Fehlerbereich, innerhalb dessen das Ergebnis unserer Vergleichsmessungen — $\frac{D_{PK}}{D_{ZK}} = 0.993 = V$ — garantiert werden kann, ergibt sich folgendermassen: Der prozentuale Fehler von V ist gleich der Summe der proz. Fehler der Faktoren auf der rechten Seite der Formel von S. 00: $F_v = 2\,F_{\Delta U} + F_{LZ} + F_{LP} + F_K$ (der proz. Fehler der Werte $(\Delta U)_{PK}$ und $(\Delta U)_{ZK}$ war der gleiche). $F_{\Delta U}$ betrug bei je 5 Messungen ± 0.15 %; die max. Fehler der Bestimmung der Elektrodenlängen waren: $F_{LZ} = \pm 0.1$ %, $F_{LP} = \pm 0.2$ %, also: $F_V = \pm (0.6 \pm F_K)$ %.

F_K lässt sich auf ± 0.1 % schätzen; denn es besteht eine sehr gute Übereinstimmung der Küstner'schen Werte für K_{Str} mit den Werten, die man nach einer zuerst von BERG, SCHWERDTFEGER und THALLER gegebenen Näherungsformel (5) und der Compton'schen Formel für die Abhängigkeit des Streukoeffizienten von der Wellenlänge berechnen kann. Der Ionisationszusatz I_{Str}, der durch die im wirksamen Kammervolumen absorbierte Streustrahlung zustandekommt, ist: $I_{Str} = \sigma \cdot W_m \cdot I_p$. Dabei bedeutet σ den Streukoeffizienten der Luft, der die klassische und die Compton-Streuung enthält, W_m die mittlere Länge der Strecken, die die gestreute Strahlung innerhalb des wirksamen Kammervolumens zurücklegt, und I_p die Ionisation durch primäre Röntgenstrahlung. Danach ergibt sich mit $W_m = 26$ cm (vgl. S. 178) bei Wellenlängen oberhalb 0.4 Å für das Verhältnis $\frac{I_{Str}}{I_p}$ ein Wert von 0.7 % bzw. für den Korrekturfaktor: $(K_{Str})_{ZK} = 0.993$ statt 0.992, wie auf S. 178 aus den Küstner'schen Werten abgeleitet wurde. Es beträgt demnach insgesamt der Fehler des von uns gemessenen Dosisquotienten nicht mehr als $F_V = 0.7$ %.

Für die absolute Dosisbestimmung in »Röntgen« ist der maximale Fehler natürlich grösser. Es gilt (bei Messungen nach Townsend): $F_D = F_{\Delta U} + F_C + F_q + F_L + F_K$ (Bedeutung der Buchstaben vgl. S. 166 u. 176). Die Beträge, die diese Einzelfehler bei den in Ockstadt gegebenen Verhältnissen annehmen, sind in Tab. 6 zusammengestellt. Bei einer Eichung oder einem Kammervergleich müssen ausserdem noch gewisse »Übertragungsfehler« berücksichtigt werden (Reproduzierbarkeit des Monitorgerätes, Ungenauigkeit bei der Fixierung des Messortes), deren Grösse von Fall zu Fall abzuschätzen ist.

Aus den in dieser Arbeit beschriebenen Vergleichsmessungen lässt sich folgender Schluss ziehen: Auf Grund unserer Ergebnisse kann als bewiesen angesehen werden, dass ein prinzipieller Unterschied zwischen den verschiedenen Messystemen der Zylinder- und der Platten-Fasskammer nicht

Tabelle 6

Fehlerzusammenstellung für die beiden Standardanlagen zur Dosismessung in »Röntgen« am Max-Planck-Institut für Biophysik, Frankfurt (Main) — Ockstadt

Art des Fehlers	Hartstrahl-Eichstand ± %	Weichstrahl-Eichstand ± %
$F_{\Delta U}$	0.1	0.1
F_C	0.1	0.1
F_q	0.2	0.2
F_L	0.1	0.2 ÷ 0.5 (je nach Elektrodenlänge)
F_K	0.5 ($K = K_D \cdot K_{Sch} \cdot K_{Str}$)	0.5 b. Grenzstr. bis 0.9 ($K = K_D \cdot K_{Sch} \cdot K_{Str} \cdot K_F$)
Summe	+ 1.0 %	± (1.1 ÷ 1.8) % (je nach El.länge u. Qual.)

Besonderer Wert ist auf die dauernde Kontrolle der Kapazitätswerte zu legen. Der in der Tabelle angegebene geringe Wert für F_C gilt — nach unserer Erfahrung — nur bei Verwendung von Festkondensatoren, mit Bernstein isoliert und mit Luft als Dielektrikum, während man bei Kapazitätsvariatoren nach Harms nach längerem Gebrauch mit etwas grösseren Fehlern rechnen muss.

besteht. Ausserdem ist gezeigt, dass durch die beiden in Ockstadt aufgebauten Dosismess-Standardanlagen eine einwandfreie Verifizierung des internationalen »Röntgen« in dem ganzen Bereich zwischen 3 (vgl. (6)) und 300 (vgl. (17)) kV Erzeugungsspannung auch in Deutschland wieder gewährleistet ist.

Den Herren Dr. Dorneich, Dr. Pohlit und Lang möchten wir für ihre Mitarbeit bei den Messungen unseren besten Dank aussprechen. Der Deutschen Forschungsgemeinschaft sei für die Unterstützung unserer Arbeiten besonders gedankt.

ZUSAMMENFASSUNG

In Ergänzung zu der bereits in den letzten Jahren im Max-Planck-Institut für Biophysik, Frankfurt (Main), errichteten Standardanlage zur Absolutmessung des internationalen »Röntgen« im Gebiet zwischen 40 und 300 kV Erzeugungsspannung mit zylindrischer Fasskammer wurde nun auch eine Standardanlage für das Gebiet der weichen und sehr weichen Röntgenstrahlen (5 bis 50 kV) aufgebaut. Die Weichstrahl-Standardkammer hat Parallelplatten-Elektroden und wurde in Bezug auf folgende Einflüsse genauer untersucht: Breite der Messelektrode, Breite der Luftspalte zwischen Mess- und Schutzelektrode, Länge und Breite der Schutzelektrode, Abstand der Potentialführungsfäden, Wellenlängenabhängigkeit oberhalb 50 kV. Beide Systeme wurden am Hartstrahl- und am Weichstrahl-Eichstand (bei 50 bzw. 30 kV) bei weitestgehender Gleichheit aller übrigen Messbedingungen miteinander verglichen und das Dosisverhältnis zu 0.993 ± 0.007 gefunden. Beide Kammertypen gewährleisten also sowohl prinzipiell als auch in ihrer hier vorliegenden praktischen Ausführungsform die Verifizierung des internationalen »Röntgen«. Die Fehlermöglichkeiten sowohl bei den Vergleichsmessungen wie auch bei den absoluten Dosismessungen an beiden Eichständen werden im einzelnen diskutiert.

SUMMARY

The standard apparatus with a cylindrical tube chamber, erected in recent years at the Max Planck Institute for Biophysics, Frankfurt (Main), for the measurement of the international roentgen in the 40—300 kV range, has now been supplemented with a standard apparatus for soft and very soft roentgen rays (5—50 kV). The soft ray standard chamber has parallel plate electrodes and was studied for the following influences: breadth of the measuring electrode, breadth of the gap between the measuring and the guard electrode, length and breadth of the guard electrode, distance between the potential leads, and energy dependence above 50 kV.

The two systems were compared with each other at 50 and 30 kV under conditions otherwise as identical as possible, and the dose relationship was found to be 0.993 ± 0.007. Thus in principle as well as in practice, these two types of chambers are in conformity with the international roentgen.

The sources of error in the relative measurement and in the absolute measurement of the dose with both chambers are discussed in detail.

RÉSUMÉ

L'appareil étalon à chambre cylindrique construit dans ces dernières années à l'Institut Max Planck de Biophysique, à Francfort-sur-le-Main, pour la mesure du roentgen international dans l'intervalle 40—300 kV a maintenant été complété par un appareil étalon pour les rayons mous et très mous (5—50 kV). La chambre étalon pour rayons mous a des électrodes planes parallèles et a été étudiée pour les influences suivantes: largeur de l'électrode de mesure, largeur de l'espace entre l'électrode de mesure et l'électrode de garde, largeur et longueur de l'électrode de garde, distance entre les conducteurs de potentiel et dépendance d'énergie au-dessus de 50 kV.

Les deux systèmes ont été comparés l'un avec l'autre (respectivement à 50 et à 30 kV) dans des conditions par ailleurs aussi identiques que possible, et le rapport de doses fut trouvé égal à 0.993 ± 0.007. Ainsi, en principe comme en pratique, ces deux types de chambres sont en conformité avec le roentgen international.

Les sources d'erreur dans la mesure relative et dans la mesure absolue de la dose avec les deux chambres sont étudiés en détail.

ZUSATZ ZUR KORREKTUR

Eine genaue Ableitung und Analyse der Grundformel für die Dosisberechnung findet sich in einer Arbeit, die in Kürze in Buchform erscheinen wird:

Rajewsky, Bunde, Dorneich, Lang, Sewkor, Jaeger u. Hübner: Darstellung, Wahrung und Übertragung der Einheit der Dosis für Röntgen- und Gammastrahlen mit Quantenenergien zwischen 3 und 400 keV. Richtlinien zur Standarddosimetrie.

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BOOK REVIEW

MEDICINA NUCLEARE. Introduzione alle applicazioni medico-biologiche della fisica nucleare. — By Aldo Perussia and coll. Pp. 887, Figs. 143, Tables 23. Il Pensiero Scientifico Ed., Rome 1954. Italian Lira 7,000.

The book is divided into four parts.

The first part summarizes the basic principles of nuclear physics (in collaboration with C. SALVIETTI), general radiobiology, dosimetry, and radiochemistry (in collaboration with L. MALATESTA). The basic conceptions of the artificial production of radiation (roentgen installations and large accelerators, in collaboration with E. GATTI), nuclear reactors, the production of radioisotopes (in collaboration with M. SILVESTRI), and the general biologic behaviour of radioisotopes (stable and unstable) are reviewed.

The second part of the book deals with instruments, techniques, and methods employed, in the analysis of radiation. In the chapter on instruments, written in collaboration with E. GATTI, apparatus for physical measurements (based on the phenomena of ionization and excitation) as well as chemical apparatus (utilizing the secondary phenomena of energy absorption processes, *i. e.* the chemical modifications of the substance irradiated) are described. In the chapter on techniques and methods (in collaboration with U. FACCHINI) an account is given of ionometric methods (condensation chambers and integrating chambers), of counter techniques (with Geiger-Müller counters), scintillation counters, and photographic methods.

The application of radioisotopes in biologic research, as well as clinically, are dealt with in the third section of the work. Following a discussion on general methodologic principles, the reader is reminded of the more important directions of research and certain practical clinical results. Mention is made of the use of radioisotopes in biochemical analyses (the reversibility of an enzymatic reaction, identification of intermediary compounds, diluted isotopes, differences in the quantitative dosage of a compound, etc.) in studying the dynamics of biologic processes and in biologic research *in vivo* (absorption, assimilation, excretion, distribution of isotopes, etc.). Certain techniques which seem to offer very promising diagnostic possibilities are described in detail (localization of tumours, evaluation of the endocrine functions of the thyroid, the hemopoietic activity, etc.).

The fourth part of the book deals with the various possibilities and modalities for the application of nuclear physics in therapy; the use of high energy electronic roentgen radiation (radio-iodine in hyperthyroidism and carcinomatous metastases of the thyroid, radio-phosphorus in the treatment of lymphopathy, radio-colloids, etc.) are discussed in some detail. Theories concerning the use of heavy particle radiation and neutron radiation, the practical application of which still seem rather distant, receive but brief attention.

The biologic risks connected with the exposure of personnel to ionizing radiation form the subject of an appendix. Emphasis is placed on the conception of 'dose tolerance' and standardized means of protection.

In view of the complexity and broadness of the subject matter treated, any discussion of the details of this work would demand the attention of a great number of specialists. It may be stated, however, without any reservation whatsoever, that the book is extraordinarily well put together and represents the answer to a long-felt need. In years to come it will certainly constitute an essential volume in the library of a progressive physician.

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